THE PRACTICE OF VETERINARY MEDICINE

458

BY

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WITH ONE HUNDRED AND SIXTEEN ILLUSTRATIONS

Sixth Revised Edition

BASIC AGRICULTURAL TRAINING SCHOOL
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PATTUKKOTTAI

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PREFACE TO THE SIXTH EDITION

During the past few years progress in the control of animal disease has surpassed that of any comparative period in the history of veterinary medicine. This has been chiefly due to advancement in the knowledge of biochemistry, and especially to the discovery of penicillin by Fleming in 1928. By 1951 antibiotic production was the most important single operation in the pharmaceutical industry in the United States. Perhaps its chief influence in veterinary medicine relates to diseases caused by various specific or nonspecific infections in which the bacterial cause is unknown, or in doubt, or in controversy, such as calf pneumonia, calf scours, and diseases of the newborn in general; also mastitis, pneumonia, and undiagnosed febrile diseases. In many instances the traditional need for determination of the bacterial cause, the pathology, and even the diagnosis, has been by-passed by the curative effect of this new remedy. Its effect is claimed to be the cause of prolongation of life in man. By-products of manufacture of antibiotics have reached the bins of the feed-stores, as a stimulant to growth and a protection to the health of the young. An increasing interest in these products is reflected in the agricultural press where the space devoted to the health of animals was never so great. The problem of animal industry is gradually shifting from survival to surplus, and its immediate future will present a lively horizon.

Because of the shifting interest and importance of various diseases of animals, brought about by the passing of the horse, and the virtual control of certain specific diseases, such as tuberculosis, the revision of a text on veterinary medicine brings to one's attention the rapidly shifting importance of the various subjects in the table of contents. Just where to omit the old and begin the new is difficult to estimate. In this respect the author has taken a middle position by postponement of omissions and an effort to keep up with the new; the past has not yet completely passed, but it definitely has changed. Among the new topics are leptospirosis, atrophic rhinitis in swine, scrapie, bluetongue, transmissible enteritis in baby pigs, displacement of the abomasum, molybdenum poisoning, soybean meal poisoning, insecticides, and a table of the normal cellular and chemical constituents of the blood.

With each new edition, changes in bacteriological terminology contribute to an increasing confusion which sometimes approaches chaos. In this respect preference has been shown for the use of established names. A change in the name that a bacterium has carried for genera-

tions, as *Escherichia coli* for *Bacterium coli,* for no obvious reason, suggests that the terminology of the subject is out of control.

For assistance and suggestions, my sincere thanks are expressed to the staff of the New York State Veterinary College; to Dr. James Baker on leptospirosis; to Drs. Fincher, Fox, Roberts, and Johnson for information on many subjects and especially on methods of treatment, diagnosis and control; to Dr. John Bentinck-Smith for preparation of the table on constituents of the blood; to R. H. Udall for a contribution on ketosis; to Prof. D. L. T. Smith for the use of his report on salt poisoning; to the Staff of the Flower Library, and to Mrs. Betty Reichert for her skill in the location of numerous errors in the final proof.

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May, 1954.

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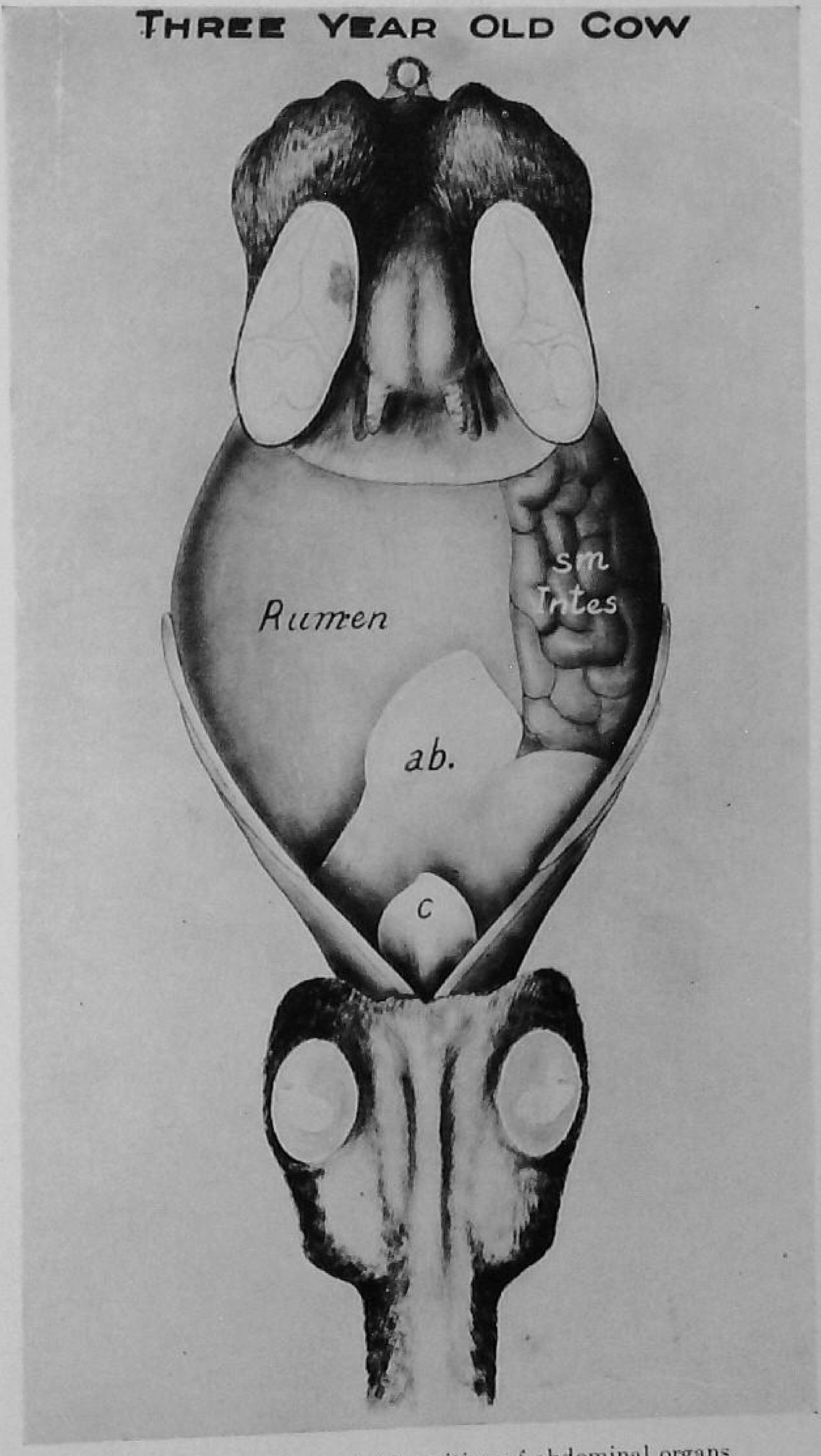
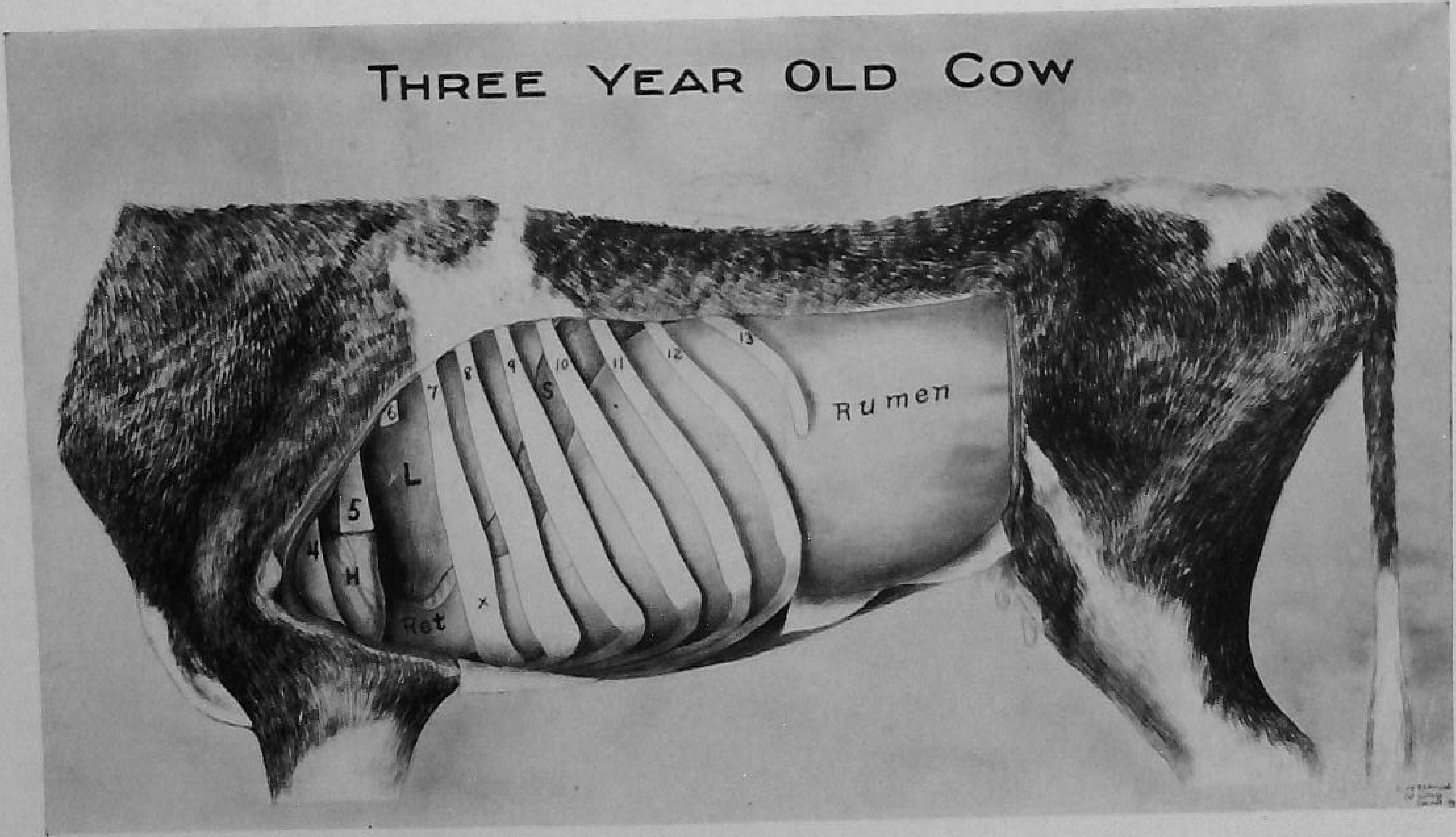
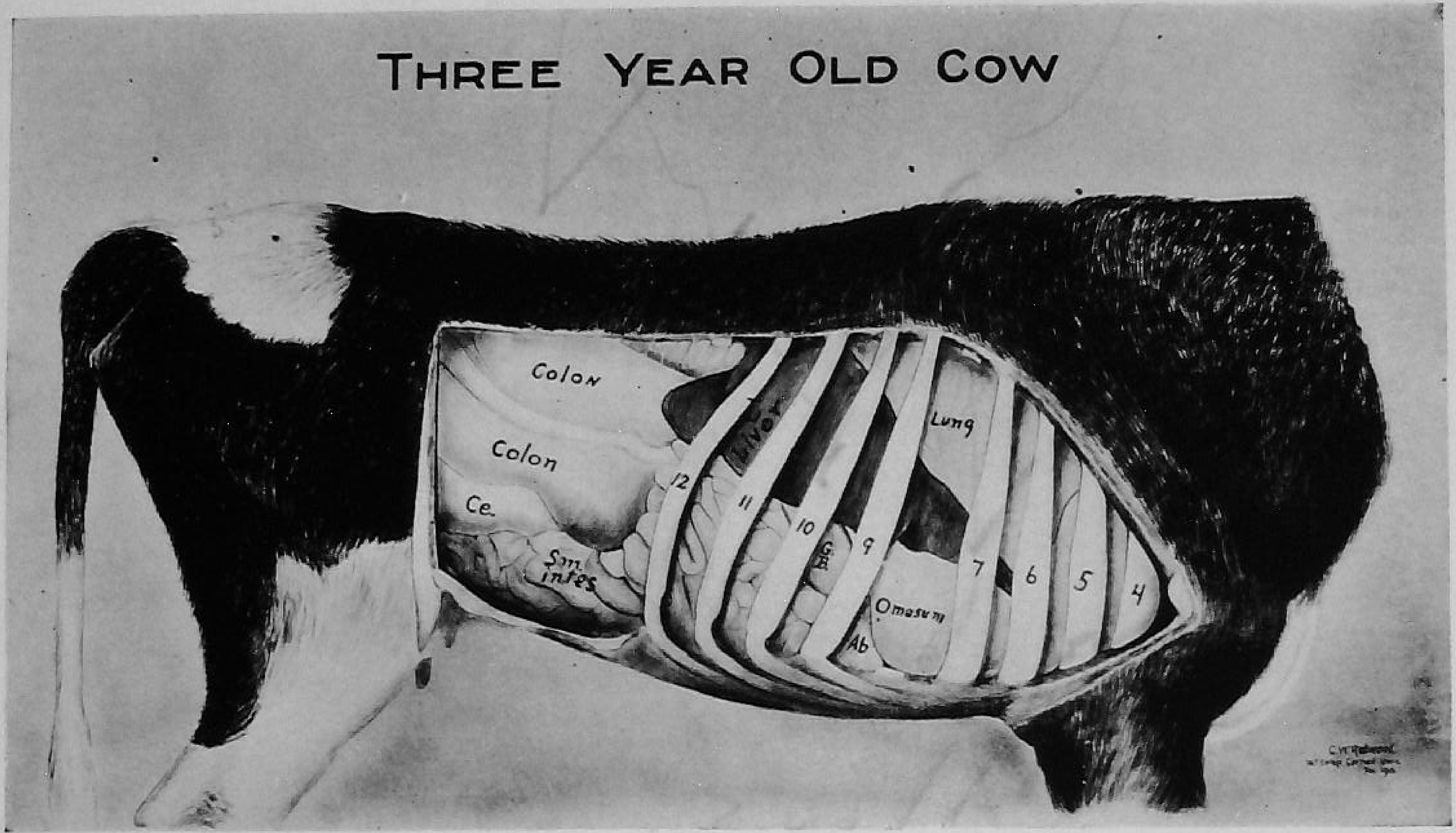


Ventral surface. Superficial position of abdominal organs.
ab, abomasum; c, xiphoid cartilage.



H, heart; L, lung; Ret, reticulum; S, spleen.



G.B., gall bladder; Ab, abomasum.

DISEASES OF THE RESPIRATORY SYSTEM

EPISTAXIS

(Nosebleed)

Etiology.—Among *local causes* are injuries from the stomach tube, contusions, fractures of the facial bones; the presence of twigs or sticks or other foreign bodies or new growths in the nasal passages, nasal catarrh (strangles, summer snuffles in cows), and erosion of the blood vessels in chronic ulcers (glanders). In fracture of the base of the skull there may be hemorrhage from both the nose and the ears with paresis. Nosebleed has been observed in severe infectious rhinitis in swine and sheep and in heavy infection with *Oestrus ovis* in sheep. An enzootic of sneezing and nasal hemorrhage in a herd of pigs affected with facial deformity has been described by York;[1] the hemorrhage in these pigs was due to infectious atrophic rhinitis. In a cow affected with recurrent and abundant hemorrhage from both nostrils, a hemangioma was found in the wall of the pharynx. Among *general causes* are general infections, intoxications, and poisons. The most important of these are anthrax, blackleg, septic metritis, purpura, infectious equine anemia, sweet clover disease, mercurial poisoning, bracken poisoning, and chronic copper poisoning. It is sometimes observed in heat stroke and it may result from pressure on the jugular vein by an ill-fitting collar. In 22 cases of nosebleed in the horse reported by Wirth,[2] 6 were caused by traumatism, 7 were listed as congenital, and 7 were due to internal causes. In the pedigree of a race horse that bled repeatedly from the nose without apparent cause, bleeders appeared twice. Nosebleed is a common symptom in chronic dilatation of the heart.

Symptoms.—Since blood at the nostrils may come from any part of the respiratory system, its presence alone may not indicate the source. Droplets or a thin stream of blood in race horses, after return from the track, is caused by overexertion. And similar bleeding may follow other violent movements, as in colic, as well as injuries to the face or the nasal mucosa. Slight persistent bleeding from erosion of a small vessel may result from tumors and chronic inflammations. Abundant free hemorrhage from both nostrils suggests erosion of a large blood vessel, as from a strangles abscess in the guttural pouch involving a branch of the inner carotid. Bleeding from the nose, combined with stumbling and weakness under exercise in a horse, indicates chronic heart dis-

ease; one finds palpitation of the heart and a fast weak pulse. Trickling of dark red serum from the nostrils is sometimes observed in septicemia (septic metritis, anthrax), especially in cows. Trickling of a little clear or straw-colored fluid from the nostrils of a horse suggests infectious equine anemia. Swallowing movements with nosebleed suggests pharyngeal hemorrhage. Blood mixed with foam comes from the lungs, though pulmonary hemorrhage may not show foam if the patient is at rest. Mercurial poisoning causes both epistaxis and hemoptysis. Bleeding from the nose in cows, if at all abundant or mixed with foam, indicates hemoptysis and the cause is usually pulmonary abscess; bloody fluid and foam may come from both nostrils and mouth. A cough suggests hemoptysis. Unilateral bleeding is usually of nasal origin, while bilateral bleeding usually has its origin posterior to the nasal passage. Gastric hemorrhage (hematemesis) is of doubtful occurrence in herbivorous animals and swine.

Treatment.—Severe bleeding from a local injury may be controlled by means of an adrenalin tampon. Before packing the nostrils it may be necessary to insert a tracheotomy tube. It is doubtful if the local application of an astringent, such as tinct. ferri. chlor., or tannic acid, is of material value. Bleeding from small vessels may be controlled by irrigation with a warm solution of creolin. Hemorrhage from overexertion soon stops when the patient comes to rest. Epistaxis is rarely fatal, though erosion of a large vessel in the guttural pouches or lungs may cause rapid death. Adrenalin, blood serum, blood transfusion, pituitary extract, and camphorated oil have been recommended, and they may be of some value when the source of persistent bleeding cannot be found. Marcenac[3] has reported good results in nosebleed in race horses with a slow intravenous injection of 100 cc. of a 25 to 30 per cent solution of sodium citrate. Pituitrin is said to diminish hemorrhage by increasing the coagulability of the blood. Lahiri[4] has described a case of "epistaxis" in a bull with recovery after the use of atropine; probably this was a case of hemoptysis. According to the theory presented in a discussion of this report, atropine decreases the tonicity of the vagus nerve and leads to increased blood calcium.

References

1. York, W. K., Fort Dodge Rev., 1941, vol. xii, No. 2, p. 18, abs. Vet. Bull. **12**, 145.
2. Wirth, Tierheilkunde und Tierzucht, Berlin, Urban & Schwarzenberg, 1930, vol. 7, p. 352.
3. Marcenac, Bull. Acad. vet., France, 1934, **7**, 67.
4. Lahiri, B. M., Discussion of the treatment of epistaxis with atropine sulfate, Indian Vet. J., 1938, **14**, 397.

CORYZA

(Acute Catarrhal Rhinitis; Nasal Catarrh; Cold)

Definition.—An acute infection of the mucous membranes of the nose, often involving all of the upper air passages, such as the pharynx, larynx, and larger bronchi. Acute inflammation of the upper respiratory tract often occurs in association with pneumonia, the entire respiratory system being involved.

Etiology.—Colds are often enzootic among horses in the damp months of the spring and fall, as well as in horses and other animals at exhibitions, such as state fairs. Enzootics of purulent rhinitis are not infrequent in foals and yearlings on breeding farms, where one attack confers immunity. It is possible that in these outbreaks one is dealing with the virus disease, enzootic laryngotracheitis of horses—see infectious bronchitis of equines. It occurs frequently as a limited enzootic among cows in summer. In colds, infection is always present and the disease may spread by contact. Storm and cold, fatigue, poor ventilation, sudden chilling, or exposure when warm are frequent causes. Severe purulent coryza may develop in cows in damp cold quarters; this has been observed in poorly ventilated stables where the walls were of concrete and dripping with moisture. In bovines nasal catarrh may occur in association with necrotic stomatitis or other severe mouth lesions and it may be caused by twigs that sometimes lodge and break off in the nostrils. Calves and pigs often suffer from outbreaks of colds in the form of a stable infection. These epidemics are favored when the atmosphere is cold and damp or close and sultry or when the stable is overcrowded or when there is frequent removal of older animals for replacement by those of a younger age. In calves there is an age susceptibility to colds and other respiratory diseases; this is distinct at the age of three to four weeks, increases to about six weeks and then gradually declines. Individuals of susceptible age are constantly present where there is frequent removal of older calves to make room for the newborn. It is better to keep groups of approximately the same age by themselves. In piggeries failure to provide adequate cleanliness and ventilation is a frequent cause of colds and other respiratory diseases.

Little is known of the bacteriology of colds. Jones and Little[1] have described an outbreak in calves due to *Pasteurella multocida*. Acute nasal catarrh also occurs in several of the acute general infections, such as influenza, enzootic laryngotracheitis, and strangles; it is also secondary to dental caries and alveolar periositis. Heaves, empyema of the guttural pouches, and other chronic affections of the respiratory system

cause frequent attacks of colds. As in persons, however, outbreaks occur in animals regardless of all precautions. Colds are common in all species following transportation.

Symptoms.—Cough and nasal discharge are the usual distinctive signs. There may be dullness and a moderate rise in temperature. Other symptoms are increased frequency in breathing, harsh bronchial sounds, and a loss in condition. Aside from dullness and congested mucosae, general symptoms are usually absent, though calves may carry a temperature of 103° to 105° F. The course is from one to three weeks and the cough may persist after all other symptoms have passed; this is especially true of colds in the horse where there is a tendency to localization in the larynx.

In cows, epidemics of rhinitis are occasionally observed in which there is an abundant mucopurulent exudate hanging from the nostrils in long shreds, a poor appetite and diminished milk flow. Cough may be absent and the temperature normal. The course is from a week to ten days.

Colds should not be lightly judged. In the young they may be symptomatic of dangerous acute infections, as *enzootic pneumonia*, or a persistent debilitating bronchitis. A history of a cold is frequent in tuberculosis, heaves, and other chronic affections of the respiratory tract, where it is merely secondary. A cold may be the beginning of a serious pneumonia. In general it signifies the presence of infection, and in the young it suggests faulty hygiene. In cattle, severe coryza may resemble a mild form of malignant head catarrh. In horses, it may be difficult to differentiate between epidemics of colds due to unknown infections, and mild forms of influenza, strangles, or enzootic laryngotracheitis.

Treatment.—Fresh air, with protection against drafts, chilling, moisture, filth, and fatigue is of first consideration. If the animal is in a close stuffy stable in warm weather, removal to a shed open on one side is useful. Disinfection of stables, or removal to dry quarters, often brings rapid improvement, especially to calves and pigs. Steam inhalation from a pail of water containing an ounce of creolin or a similar disinfectant affords relief; this is useful when a swollen mucosa and accumulation of exudate are causing stenosis. Pillers[2] reports beneficial effect from a single inhalation of one dram each of menthol and thymol in a quart of hot water, but for a routine measure he prefers a medicated emollient applied around the inside of the nostril. Steam inhalations are disfavored by some on the theory that the change-over from the atmosphere of hot steam to that of the average stable, when the inhalation is taken away, is too sudden and liable to worsen the condition.

Atropine sulfate 1/4 grain (.015 Gm.) two or three times daily affords relief. Expectorants and stimulants as represented in the following combination are useful:

* ℞	Ammonium chloride	
	Ammonium carbonate	aa ℥ iv (120 Gm.)
	Camphor	℥ i (30 Gm.)
	Syrup	Cong. i (4000 cc.)

M. Sig.—One-half to one ounce (15-30 cc.) three to six times daily.

An irritating cough may be relieved by including an ounce of F.E. belladonna in a pint of this mixture. Sulfamerazine (6 Gm. per 100 pounds body weight daily) administered in two divided doses is usually followed by a drop in temperature and apparent improvement. Penicillin or other antibiotics as recommended in pneumonia are especially indicated in severe attacks with possible complications. Wirth and Diernhofer[3] report relief from the symptoms of acute nasal catarrh in pigs from injection of 2 cc. 1:1000 adrenalin solution in 8 cc. warm water into the nostril with a 10 cc. syringe. The pig is raised so that the hind feet just touch the ground and the head hangs free; the end of the syringe should be short and blunt. As a protection against exposure to cold and damp floors, the raising of calves on wire netting has been recommended by Bartlett.[4]

References

1. Jones, F. S., and Little, R. B., An epidemiological study of rhinitis (coryza) in calves with special reference to pneumonia, J. Inf. Dis., 1922, **36**, 273.
2. Pillers, A. W. N., Remarks on the clinical aspects of contagious nasal catarrh in horses, Vet. Rec., 1934, **14**, 1153.
3. Lehrbuch der Innern Krankheiten der Haustiere, 1950.
4. Bartlett, J. W., and Tucker, H. H., Raising calves on wire floors, New Jersey Agr. Exp. Sta. Cir. 372, May 1937, New Brunswick.

CROUPOUS RHINITIS

As the name indicates, this is a croupous or fibrinous inflammation of the nasal mucosa. In cattle it is one of the prominent features of malignant head catarrh. Occasionally it is met with in cattle as an independent infectious disease, which may be sporadic or endemic. Outbreaks in the horse have also been described, but in all species

* Because of numerous inquiries on how to fill this prescription, the following directions are appended: Add 6.5 pounds of sugar to one-half gallon of camphor water. Shake well. Add camphor water to make one gallon. Place the entire gallon bottle in water that has just been brought to a boil, and let stand until the sugar has dissolved (three to four hours). Add the ammonium chloride. Add the ammonium carbonate after it has been triturated.

the disease is infrequent. Grunth has published a report on its occurrence in the vicinity of Copenhagen, Denmark, where it is brought to the herd in cows purchased in the public markets and is confined to cows that have recently freshened. Uncomplicated forms are characterized by croupous rhinitis and a high fever. Complications in the form of pneumonia, diarrhea and metritis are frequent and the loss is heavy. The cause is attributed to some unknown infection. It is an open question whether or not croup, as described by Grunth,[1] is a form of malignant head catarrh; there is no involvement of the brain and usually none of the eyes, and it is milder than malignant head catarrh. Croupous rhinitis is also caused by intense irritation from fumes, fire, and smoke.

The onset is sudden, with marked general symptoms, a high fever, and a fibrinopurulent discharge from the nose. The nasal mucosa is swollen, and sprinkled with petechiae. Often there is a bronchitis, and there may be enteritis with diarrhea. The enzootic form is rather fatal while the sporadic form varies widely in intensity. The treatment is symptomatic. Steam inhalations are beneficial, and the sulfonamides may be useful.

Follicular rhinitis is a granular inflammation of the mucosa resulting from other forms of inflammation, such as strangles, or croupous rhinitis.

Reference

1. Grunth, Über den Croup der Rinder, Zeit. f. Tiermedizin, 1905, **9**, 232.

PURULENT RHINITIS

Two cases of fatal purulent rhinitis in 10-day-old calves have been described by Schofield.[1] The attack was ushered in with dullness, recumbency, head drawn to one side, and incoordination of movements when aroused. When standing the head was held low and constantly moved from side to side with a weaving motion. The nasal discharge was at first slight, but became abundant and purulent after two or three days. The temperature was never elevated more than one degree. On autopsy the medial turbinate was found to be filled with a thick purulent exudate associated with extensive congestion. In one, the fluid in the cerebral ventricles was increased. Cultures from both gave a pure culture of *Corynebacterium pyogenes*.

Purulent rhinitis with multiple abscess formation in the floor of the nasal cavity has been observed in navel-ill in a week-old calf; the symptoms were severe inspiratory dyspnea, a copious flow of pus from both nostrils and swelling of the umbilicus. Purulent rhinitis has also been observed in calves after their heads were forcibly held in

milk while being "taught" to drink. This condition was complicated with purulent sinusitis, purulent involvement of the eyes, and sometimes pneumonia.

REFERENCE

1. Schofield, F. S., Report of the Ontario Veterinary College, 1936, p. 17.

CHRONIC NASAL CATARRH

Chronic nasal catarrh in the horse is nearly always secondary. It is observed in sinus infections, alveolar periostitis, and in chronic lung disease (heaves, glanders, tuberculosis).

In cows there is a primary chronic rhinitis which is rather prevalent in New York State, where it is commonly known as *"summer snuffles"* or *"hay fever."* It affects certain individuals only in the summer months, and reappears in the same animal each year when turned to pasture. The cause is unknown, though its seasonal resemblance to hay fever suggests a similar etiology. As a rule only one animal in a herd is affected. In a mild form it only causes an inspiratory snoring sound that may be heard at some distance. In more severe forms there is a bilateral purulent discharge from the nostrils; this may so completely occlude the nasal passages that mouth breathing occurs. At times several ounces of yellow pus may be forcibly expelled. In some there is also nosebleed. Lachrymation is common. The cow may rub the nose on the fence or on a stub, and in this manner force the stub into the nostrils for several inches, where it may break off and require removal. Difficult breathing with distress on exercise is uniformly present. The appetite and condition remain unchanged, but the milk flow is often reduced. When the animal is stabled in the fall, recovery follows, but the rhinitis recurs during the next season at pasture. Treatment with drugs and nasal irrigation has not usually been effective, but steam inhalations and atropin may afford temporary relief. Improvement occurs if the patient is stabled.

A few cases of persistent rhinitis in cows, combined with clonic spasms of the neck muscles, forced movements, and other symptoms of motor irritation, with final recovery, have been observed. While the diagnosis was in doubt, they were suggestive of a mild form of malignant head catarrh. Apparent benefit resulted from irrigation of the nostrils with either a weak chlorine or iodine solution. In persistent chronic nasal catarrh in cows characterized by an abundant purulent nasal discharge and associated with involvement of the bronchi, intravenous injection of sodium iodide (1 ounce in 500 cc. distilled water), one injection, may be beneficial. Sulfamerazine or penicillin may be useful.

ATROPHIC INFECTIOUS RHINITIS IN SWINE

Definition.—Infectious atrophic rhinitis in swine is an incurable chronic contagious affection of obscure pathology and unknown etiology affecting chiefly the young and characterized by sneezing, nosebleed, stunted growth, and dissolution of the turbinate bones. Increasing prevalence is reported from Canada and the Midwest where it is a comparatively new disease which interferes seriously with the growth of affected pigs and the sale of stock from breeding herds.

It is probable that the disease described in Sweden and in Germany under the name Schnüffelkrankheit (snuffling disease) and Niesenkrankheit (sneezing disease) is identical with the atrophic rhinitis of swine in America. In the earlier European descriptions the etiology was ascribed to various causes, as hereditary weakness, hormone deficiency, mineral imbalance, secondary infection, etc., but in 1940 Thunberg and Carlstrom[1] demonstrated that it is contagious.

Etiology.—In 1944 Doyle and associates[2] wrote that "during the past three years we have seen a dystrophic rhinitis in five herds in swine in as many widely separated sections of Indiana. Reports of what appear to be the same disease have come to us from several other herds. . . . It has been of great economic importance in the affected herds but has not yet become widely prevalent in the State." The disease is usually introduced into a herd through the purchase of a carrier animal. It has been spread by direct contact with infected swine, by nasal instillation of a suspension from the nasal contents of infected swine, and by exposing pigs by contact a few days after birth. Feces and urine are apparently negative. The infectious agent was not determined by Schofield and Jones,[3] either by a bacteriologic examination or by a search for a filterable virus, but control pigs receiving unfiltered emulsions developed typical lesions. Gwatkin et al.[4] have reported that instillation of washings from adult pigs with rhinitis produced a partial or complete disappearance of turbinated structure in baby pigs, the condition being fairly definite at 30 days; they were unable to infect laboratory animals; heating at 45° C. for one hour did not destroy the activity of the suspension of nasal material, but portions heated to 60° C. were inactive; filtrate, supernatant centrifuged material, and bacterial cultures were negative. In 1953 Gwatkin and associates[6] reported that "rhinitis indistinguishable from the usual experimental disease was produced in baby pigs with nasal instillation of *Pasteurella multocida*, Type B, isolated from a field case of rhinitis."

Symptoms.—Following the addition of an infected animal to a herd, a period of two to three years may pass before the disease becomes

general. Affected pigs often sneeze violently and in rapid succession; sometimes this is accompanied by jumping into the air in an effort to clear the nose. Often the sneezing is accompanied by a nasal discharge of blood, so that the floor and walls are heavily smeared. Other symptoms are constant rooting, rubbing the nose against hard objects, holding the head to one side, and coughing. In some individuals the most conspicuous symptom is a distortion of the snout. Depending on

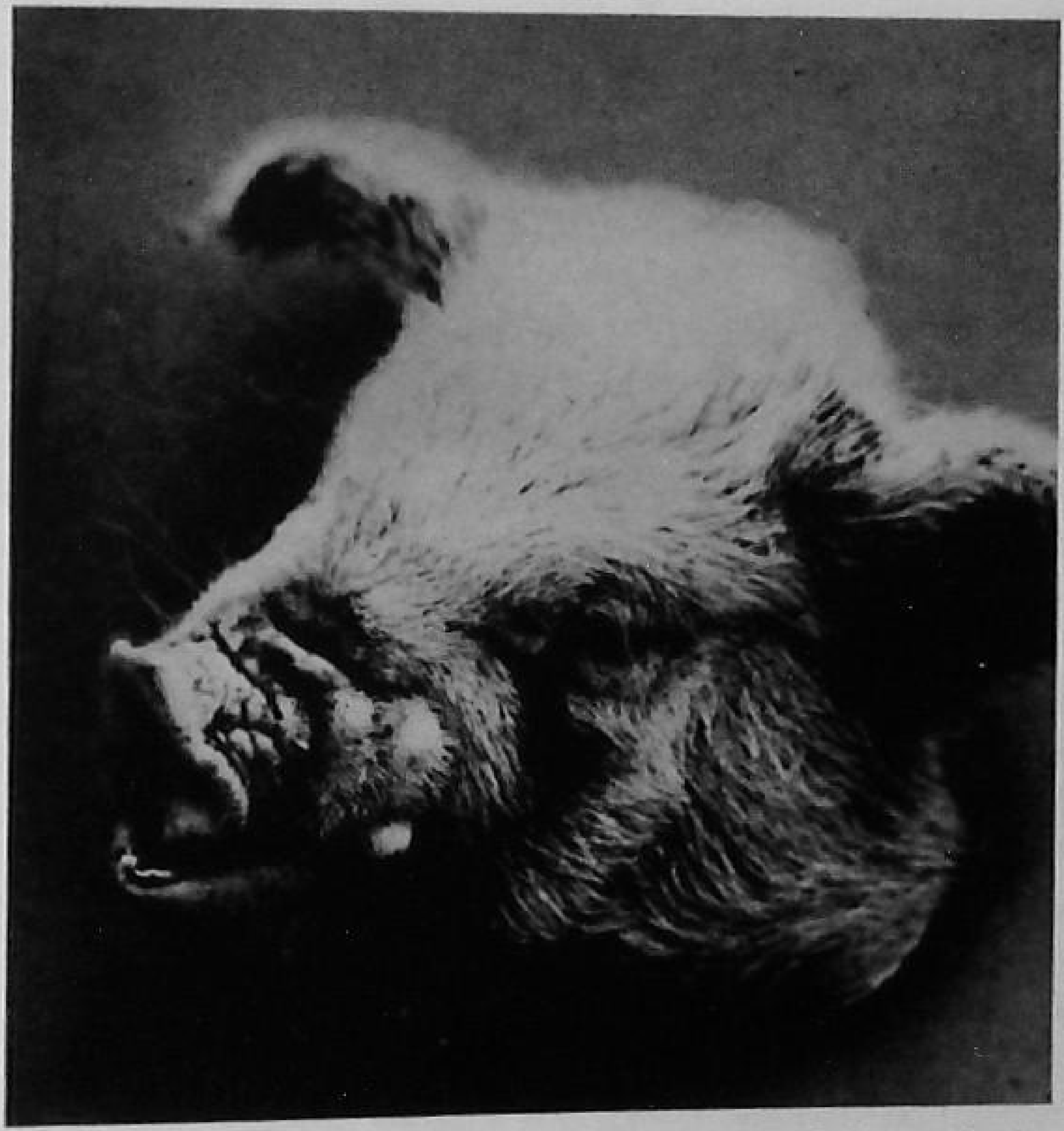


Fig. 1. Atrophic rhinitis. (Courtesy of Fort Dodge Laboratories.)

whether the atrophy of the turbinates is unilateral or bilateral the snout may be turned to one side, or upward giving the pig a dish-faced appearance; in some the snout is short and wrinkled ("bull nose"). While older swine are susceptible, it is especially noticeable about weaning time, and the chief loss is economic from stunted growth.

Morbid Anatomy.—The turbinate bones are the seat of the predominant and the characteristic lesions. As described by Schofield and Jones:[3] "In the earliest state of the disease numerous small foci of congestion are present on the external surface of the turbinates, which soon become less rigid and areas of softening may easily be penetrated by any sharp-pointed instrument. In two to four weeks the turbinates

may have disappeared and a narrow strip of firm tissue covered by mucous membrane alone remains. A mucopurulent discharge is present in the nasal passage. In many early cases, the external surface of the turbinate is practically free from any inflammatory exudate. If the bone is removed and examined, a tenacious mucopurulent discharge fills the inner and lesser curvature." In the majority of cases of advanced rhinitis the ethmoids are also involved. The direct cause of atrophy of the turbinates is undetermined, and in other forms of rhinitis severe atrophy of the turbinates is unknown. Lateral distortion of the face occurs when involvement of the turbinates is unilateral, and the face is "dished" when both turbinates are involved. In advanced cases pneumonia is a frequent complication. In experimental pigs Gwatkin et al.[4] reported loss of turbinates when no definite symptoms were noted during the period of observation and the condition was only discovered by necropsy.

Diagnosis.—Until recently there has been no clear distinction between "bull nose" and atrophic rhinitis, but it is now known that there are two separate diseases. Doyle[1] writes that "bull nose" is characterized by the presence of granulomatous swelling with more or less necrosis in the central portion, and that there is rarely any distortion of the snout other than that which results from the swelling. And Hutchings[5] reports that uni- or bilateral partial or complete dissolution of the turbinates at a point of transection half way between the end of the nose and the eyes is pathognomonic for atrophic rhinitis. In the early stages the disease closely resembles "bull nose," and a diagnosis may depend on a history of previous cases on the farm.

Control.—Since treatment is ineffective and the economic loss is heavy, it is generally advised to sell the entire herd, clean and disinfect the premises, and restock after a few months.

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INFECTIOUS RHINITIS IN SWINE

(Bull Nose; Sniffles)

Definition.—A chronic inflammation of the mucosa of the nose and adjacent sinuses, leading to deformity of the facial bones, and often associated with toxemia. It prevails chiefly in the swine-raising districts of the Mississippi Valley, where it is a frequent cause of loss among pigs. Adults are occasionally affected.

Etiology.—Rhinitis in pigs is endemic on certain farms in animals from 6 to 8 weeks of age, it seldom affects more than 10 per cent of the herd, and apparently does not spread directly from one pig to another. It is favored by the constant use of the same lots and by the mud and filth of wet seasons. According to Fitch[1] and Van Es[2] it is a soil infection with *Actinomyces necrophorus*, a form of necrobacillosis. Kinsley[3] reports that as a rule when bull nose first appears on a farm, only a few pigs are attacked, but each succeeding year the percentage of infected pigs increases and by the fourth or fifth year after the disease first occurred it is not unusual for 25 to 40 per cent of the pig crop to become affected; that it may be introduced by the addition of apparently healthy pigs from an infected farm; and that it may also develop in healthy pigs taken from a farm where there was no bull nose, and yarded in lots where cases of the disease have previously existed.

Symptoms.—The early symptoms are like those of a simple nasal catarrh. There is a slight eruption of the skin at the nostrils, and a slight serous or mucopurulent nasal discharge which may contain blood. Sneezing and snuffling are common. Sneezing and shaking the head as though trying to dislodge a foreign object are mentioned as occasional symptoms by Hastings,[4] who also reports that often the course is so gradual that the symptoms are not observed until after a few weeks when bulging of the facial bones becomes distinct—"bull nose." Poor appetite, unthriftiness, and conjunctivitis are the rule. After a time fistulae may open on the face and discharge fetid pus. Death may finally occur or the pig may survive in a stunted form. Since the discovery of the specific nature of atrophic rhinitis in swine, the relative importance of bull nose has become secondary.

Autopsy reveals extensive necrosis of the mucous membranes of the nasal passages and facial sinuses and similar changes are found in the turbinated bones and the bones of the face. Abscess formation may be found in the lymph glands of the head, and metastatic necrotic foci may be present in the lungs.

Treatment.—A prompt cure may be obtained in the first stages by dipping the snouts of the pigs in a creolin solution. When the lesions

are advanced the disease is generally regarded as incurable. Fitch has reported improvement from bull nose in a 200-pound hog by daily spraying of water over the snout. It can be prevented by cleanliness and disinfection of pens.

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ANIMAL PARASITES IN THE NOSE AND FACIAL SINUSES

(Sheep Gadfly; Oestrus ovis; Grub in the Head; Sheep Bot)

The sheep gadfly, *Oestrus ovis*, attacks pastured sheep in the heat of the day, and deposits larvae on the margins of the nostrils. The larvae migrate to the frontal sinuses where they develop, causing nasal catarrh, and in rare instances symptoms of meningitis.

While this fly is reported by Osborn[1] as the most common insect pest of sheep, its effect is not serious. It is said to occur throughout the world. The adult female is about ½ inch long, very active, and difficult to see while depositing eggs, but remaining quiet in the cool morning and afternoon. The deposited larvae are about 1/16 inch long; they have two hooks at the head, and spines on the abdomen. In the sinuses they attach themselves by the two hooks, and reach maturity in about ten months; the length is then about ¾ inch. From three to six are usually present. At maturity they emerge from the nostril, enter the ground, and at the end of about a month reappear as mature flies. Cobbett and Mitchell[2] report that in western Texas, where the winters are moderate, the larvae continue to develop in the heads of sheep and are expelled by the host animals the year around; but in New Mexico, where the winters are cold, the development of larvae in the heads of sheep is suspended in the fall and winter when the infestation consists chiefly of first-stage larvae located in the nasal passages. These minute larvae remain quiescent on the nasal mucosa and do not migrate to the frontal sinuses of the host animals for further development until the following spring and summer.

Symptoms.—When the sheep are attacked by the fly, they shake their heads, stomp, and thrust their heads to the ground; they huddle together under trees or other shelter, holding their noses under one another to prevent an attack. They may run in terror holding the nose near the ground. Migrating young larvae irritate the mucosa and cause

sneezing and other signs of irritation of the nose. In the spring, when the adult larvae loosen and pass out, the nasal catarrh is characterized by sneezing, a dirty nasal discharge, and lachrymation, yet the catarrh is seldom severe. It is claimed that in rare instances they migrate through the ethmoid sinuses, causing fatal meningitis. Severe inflammation and hemorrhage of the nasal mucous membranes, caused by massive infestation, has been described by Dill[3] in Nevada; deaths were frequent. Du Toit and Clark[4] write that on account of the entire immunity from destruction that this parasite has enjoyed it is becoming a menacing factor in the sheep farming industry in certain parts of South Africa. They found larvae of all sizes in the nasal passages and sinuses throughout the year. Sheep heavily parasitized with *Oestrus ovis* stand with the head hanging and blood-stained mucus exudes from the nostrils; in bad cases they refuse to eat. Heavy losses from gross infection with *Oestrus ovis* have occurred in South Africa.

Treatment.—Relief is best obtained by prevention. Gadflies rarely enter buildings, and if the sheep are provided with a darkened shed they may avoid attack. The noses may be smeared with tar. This is done with the use of logs containing holes 2.5 inches in diameter, bored 4 inches deep, at intervals of about 6 inches; salt is placed in the holes, and tar is smeared around them. Various inhalants, as lime, tar, benzine, sulfur, and a hot close atmosphere, have proved useless in dislodging the adult larvae from the sinuses. Gidlow and Hickman[5] have reported on the use of equal parts carbon disulfide and light mineral oil (3 cc. in each nostril). While the drug was fatal to larvae with which it came in contact, injection into the sinuses was found to be difficult. In the heads of 21 sheep, 6 of 16 larvae were destroyed by this method. In a brief comment on the preceding report, J. R. Stewart[6] of Australia wrote: "We have been treating this disease for many years with no trouble or risks. We use a 5-cc. syringe needle, 1 inch long, truncated, with a hard steel stilet inserted in the needle. The stilet has a flat head. After restraining the sheep to be treated, apply the needle and stilet to the desired spot. With a sharp blow with a wooden mallet, drive the needle into the sinus, withdraw the stilet, attach the syringe to the needle, inject 2 cc. of solution and withdraw the needle. Site of operation: For rams with horns (Merino): ¼ of an inch from the base of horn (one injection on each side). For ewes, wethers and rams without horns, inject into the frontal sinus near the median line, parallel to the supra-orbital process of the frontal bone (upper limit of the eye-socket). One injection should be made on each side of the median line. The method is quick, causes no trouble and is effective." After a study of this condition in sheep in Texas and New Mexico, Mitchell and Cob-

bett[7] concluded that application of pine tar to the nose at intervals of twice a week had little repellent effect on the adult fly. They observed pathological changes only in those cases where grubs had died within the head cavities, and they questioned the value of treatments intended to kill grubs in this location; experiments with the salt-trough method of applying repellents to the noses of sheep showed some promise of usefulness. One may trephine the frontal sinus and remove the larvae with pincers.

Du Toit and Clark[4] confirm Stewart's[6] report that destruction of the larvae in the frontal sinuses is a practical and effective method of treatment. Into each frontal sinus they inject 2 cc. of a mixture of equal parts carbon bisulfide and liquid petrolatum. Within one to two minutes the sheep recover from the intoxicating effects of the drug and within a day or two the larvae disappear from the sinuses. Du Toit writes that "in routine postmortem examinations of sheep at Onderstepoort, where the cause of death can definitely be established as something apart from *Oestrus ovis* infection, dead and often partially decomposed larvae are a fairly common finding even in the case of normal slaughtered sheep. . . . In all cases where *Oestrus ovis* was found on post mortem to be the principal parasite affecting the sheep, marked and rapid improvement followed the injection. . . . The method is not safe for sheep under 6 months of age."

Cobbett[8] reports that the first-stage larvae may be expelled from the nasal passages with a 3 per cent solution of saponified cresol (lysol) applied in the fall and winter months before they migrate to the frontal sinuses. The solution is forced into the nasal passages from tanks in which air pressure is maintained at 35 to 45 pounds, using 1 ounce (30 cc.) in each nostril. Approximately 90 per cent of infestation is eliminated by one treatment, and 98 per cent by a second treatment five days later.

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PHARYNGITIS

(Sore Throat; Angina)

Definition.—Pharyngitis in equines is an acute inflammation of the mucosa and submucosa of the pharynx, usually with abscess formation in the retropharyngeal lymph glands. To a variable extent it involves the mucosa of adjacent organs, as the nose, the larynx, the fauces and the guttural pouches. Clinically it is marked by an abundant purulent nasal discharge and by regurgitation through the nostrils when drinking.

Etiology.—In equines it is often enzootic in the fall and spring. Animals of any age are affected, but it is most common in young adults. Pharyngitis may be the result of mechanical, chemical, or thermic irritation, but in the vast majority of cases it is due to infection and is transmissible. Predisposing causes are cold damp stables, exposure to cold drafts when warm, and similar atmospheric extremes. Sometimes a capsule of creolin, chloral, or other irritant drug breaks in the throat and sets up an inflammation. It is a partial condition in strangles, and in a mild form of strangles it may be the only manifestation of the disease. Hoare[1] expresses the opinion that so-called enzootics of pharyngitis are in reality outbreaks of strangles or influenza. At the veterinary clinic in Vienna, Wirth[2] has for years conducted a routine laryngoscopic examination of the guttural pouches of horses affected with pharyngitis. These examinations show that in at least 90 per cent of pharyngitis there are abscesses in the subparotid and retropharyngeal lymph glands, and especially in those which may be designated as lymph nodes of the guttural pouches. These guttural pouch lymph nodes lie above the arytenoid cartilages between the mucosa of the guttural pouches and the posterior pharyngeal wall. Smears made from the pus of these abscesses are identical in appearance with those obtained from a strangles abscess, which leads Wirth to the conclusion that pharyngitis in the horse is nearly always strangles.

Morbid Anatomy.—The mucous membrane of the pharynx is congested, swollen and covered with mucopurulent or hemorrhagic exudate. Pus foci and ulcerative surfaces may be present. The presence of fibrinous or diphtheritic membranes is rare. In the horse the posterior wall of the pharynx may be thickened, phlegmonous, and infiltrated with pus, while the retropharyngeal lymph glands are swollen and contain abscesses.

Symptoms.—General symptoms occur according to the severity of the attack. The onset is marked by depression, partial or complete refusal to eat or drink, and a painful suppressed cough. When water is swallowed a portion of it regurgitates through the nostrils and swallowing is painful. Because of sore throat the head is carried in an extended position. Local examination reveals a purulent bilateral nasal discharge, pain and suppressed cough when the throat is pinched. The abundant purulent nasal discharge comes from the guttural pouches, where abscesses of the retropharyngeal lymph glands have ruptured. Difficulty in swallowing and regurgitation are due to paralysis of the upper wall of the pharynx which is made immobile by the presence of abscesses and edema. The temperature is from 103° to 105° F. The usual form runs a typical course, reaching its height about the fourth to the sixth day, and terminating in recovery in four to six weeks. With extensive abscess formation the breathing is stenotic and labored and there is local swelling; the course is irregular with a prolonged convalescence. Toxemia, exhaustion, and loss of condition are often present. This form may terminate in septicemia, pneumonia, or pharyngeal fistula. While complete resorption of the abscesses in the walls of the guttural pouch is the rule, they may persist for some time after apparent recovery and cause symptoms of chronic respiratory disorder. Pharyngitis due to chemical irritants, such as creolin, usually recedes within a week.

Diagnosis.—The dominant signs are bilateral purulent nasal discharge, extension of the head, pain and suppressed cough on compression of the throat, and regurgitation of water through the nostrils. In paralysis of the pharynx there are no general symptoms, pain or swelling, and the nasal discharge consists of saliva mixed with food.

Treatment.—A horse with pharyngitis should be segregated to prevent the spread of the disease. The stall should be dry and ventilated, but the animal should be protected against drafts. In cool weather the body and limbs should be clothed. The manger and walls frequently are smeared with purulent nasal discharges; all such surfaces should be kept clean and disinfected. It is advisable to keep before the sick horse a pail of water containing ½ ounce of chlorate of potash; this should be renewed often. The diet is of little consequence during the first three days, when the throat is so painful that chewing and swallowing are difficult. Linseed or oatmeal water are suitable substitutes for solid food during this period. Clean washed roots, such as carrots, may be offered frequently in small quantities. Whatever the patient desires and can eat, may be given.

The medicinal treatment is symptomatic. It is probable that the

greatest degree of relief may be obtained by steaming with creolin or similar vapors. Special steam-kettles are excellent, but are no better than a sack fitted over the steaming pail and the patient's nose. Additions of warm bricks or stones to the hot water provide continuous steam. This not only relieves the pain, but it seems to reduce the swelling. I have observed marked relief from inspiratory dyspnea within a few minutes after the steaming began. The duration of exposure to steam may be limited by the attendant's time. Severe dyspnea may be relieved by this method, thus avoiding tracheotomy. When an abscess is recognized, early incision is indicated. Heat may be applied externally. In a severe form, when pain and swelling are marked, hot poultices and antiseptic packs afford relief. Wirth[2] reports benefit from exposure to a sun lamp fifteen minutes daily, followed by a pack over the throat; after rupture of the abscesses, irrigation of the guttural pouches is advised. For this purpose he mentions a special catheter that may be easily passed after sufficient practice. European authors advise a spirits of camphor pack. Mild cantharides or mercury blisters over the throat are often used, but they are of questionable value except for circumscribed persistent swellings that remain after the acute heat and pain have passed. Stimulation, and relief from painful cough, may be obtained by the use of expectorants combined with hyoscyamus or belladonna in syrup:

℞	Ammonii chloridi		
	Ammonii carbonatis	āā ʒ iv	(16 Gm.)
	Camphorae	ʒ i	(4 Gm.)
	F. E. belladonnae	℥ i	(30 cc.)
	Syrupi q. s.	℥ xvi.	(500 cc.)

M. Sig.—One-half to one ounce (15-30 cc.) every two to four hours.

In the administration of medicine there is danger of causing inhalation pneumonia, either by drenching, or by forcing liquids from a syringe into the back part of the mouth. A safer method is to pour the liquid on the tongue from a half-ounce shell vial, or to administer it in the form of an electuary. The direct application of disinfectants to the pharyngeal mucosa by means of a special atomizer or otherwise is of doubtful value.

Fistulae of the pharynx and esophagus are successfully treated by withholding roughage, and feeding on milk, eggs, oatmeal gruel, etc. Unhealthy granulating margins may be freshened and brought together with sutures. The external wound is cleansed daily and disinfected with an astringent, such as argenti nitras 1:5000 in water.

If pharyngitis in the horse is nearly always strangles, as claimed by

Wirth, methods employed in the prophylaxis and treatment of strangles should also be useful in the treatment of pharyngitis. Penicillin has been reported by Roberts[3] and by Semtner[4] as useful in the treatment of pharyngitis.

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PHARYNGITIS OF SWINE

Pharyngitis in swine is said to occur often, yet one finds few descriptions of the disease. It has been seen only rarely in our clinic and then in association with laryngeal catarrh. Hastings[1] reports that it is important on many farms in the Middle West as a nonfatal disease of pigs from 5 to 8 weeks of age. It is manifested by cough, sometimes abscess formation in the throat region, and slight fever; the condition may persist and result in stunted growth.

The *etiology* is attributed to unsanitary quarters, damp or cement floors, dust, and infection. Anthrax in swine usually localizes in the throat causing a fatal malignant edema.

The *symptoms* of pharyngeal catarrh in swine are cough and failure to take food because of pain. Liquids are swallowed without regurgitation, but the pig may vomit. In acute cases the submaxillary region may be swollen and painful, causing the neck and head to be held stiffly. Cough is always present, is most marked when the pigs are first disturbed, and may persist for some time after other symptoms have passed. There may be severe inspiratory dyspnea with other signs of edema of the glottis. Abscesses that form in the submaxillary region rupture and discharge thin fetid pus. The affection is a nonfatal general catarrh of the upper respiratory tract which affects chiefly the tonsils and submaxillary lymph glands, or it may be a localization of a general infectious disease such as anthrax or swine influenza.

Treatment is limited to surgical care of the abscesses and correction of the sanitation.

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PHARYNGITIS OF CATTLE

Acute catarrhal pharyngitis in a mild form is occasionally seen in cattle when enzootics of respiratory affections are prevalent in the cold seasons, when cattle are housed in cold damp stables, and following exposure to storms. In such cases one observes an inspiratory stenotic or snoring sound, soreness and sometimes swelling in the pharyngeal region, and usually an induced cough. Cows do not present the same degree of nasal discharge as horses; the affection is more of the nature of an inflammatory edema. General symptoms are usually absent.

Injuries to the pharynx may result from misuse of a probang, or the balling gun in calves, or from lodgement in the throat of sharp wires or pieces of glass. In one cow, chicken wire in the pharynx caused symptoms resembling those of "summer snuffles." Depending on their extent, such injuries cause various degrees of inflammatory edema, external swelling, and inspiratory dyspnea; they heal promptly when the foreign body has been removed and when there has been no perforation. Marked inspiratory dyspnea and swelling of the throat are suggestive of injury and careful manipulation with the aid of a speculum may reveal the object. Before passing the hand through the speculum for direct palpation of the pharynx one should first exclude the possibility of rabies. A cold pack on the throat may relieve the difficult breathing.

Chronic pharyngitis has been observed in one 6-year-old cow in our clinic. There was a history of inspiratory dyspnea and snoring for several weeks, but the clinical examination was negative. An autopsy revealed inflammatory thickening of the pharyngeal wall with multiple abscesses in the submucosa. Inspiratory snoring in cows has been observed rather often as a symptom of tuberculosis of the retropharyngeal lymph glands. Yet there are other causes of chronic inspiratory dyspnea in cows, such as actinobacillosis and other unknown infections that may localize in the wall of the pharynx.

Tumors and abscess of the pharynx (peripharyngeal inflammation and tumors).—Occasionally actinobacillosis, tuberculosis, and tumors develop in the retropharyngeal tissues. The symptoms are inspiratory dyspnea with or without swelling of the pharyngeal region. In one instance of hemangioma involving the wall of the pharynx, the symptoms were repeated and profuse nosebleed, head slightly lowered, neck extended, open mouth, loud and stertorous labial breathing. When the mouth was closed air was expelled equally from both nostrils. A passed catheter met with no obstruction in the nasal passages.

Actinobacillosis in the pharyngeal wall may be difficult to locate clinically. The growths may be multiple and from ½ to 1 inch in diam-

eter. The chief symptom is inspiratory stertorous dyspnea.

Neurofibromatosis of the lungs may cause respiratory distress in cows similar to that caused by tumors in the retropharyngeal tissues.

ACUTE CATARRHAL LARYNGITIS

Etiology.—This occurs independently, in association with colds, and in the specific infectious catarrhs, as in strangles or influenza. Acute laryngitis is frequently enzootic in horses, when it spreads in the same manner as colds (enzootic laryngotracheitis). When hemorrhagic septicemia is prevalent in cattle, certain individuals may present only symptoms of laryngeal catarrh or colds. It is sometimes due to exposure to cold and wet—catching cold, for example, when cows are left at pasture in the first cold nights of the fall. Dust and foreign bodies are an infrequent cause. A severe type may result from careless use of a probang in the relief of choke or tympany.

Symptoms.—The chief symptom is a short, harsh, dry cough, easily induced. It may be frequent and severe and it is aggravated by cold air and dust. Nasal discharge and swelling of the lymph glands are infrequent. General symptoms and fever are absent except in acute febrile disease—influenza. Laryngitis in cattle is usually associated with catarrh of the trachea and bronchi. The lowered head, open mouth, and protruded tongue, peculiar to this affection in cows, sometimes lead the owner to suspect choke. Swelling of the mucosa may cause an inspiratory stenotic laryngeal sound. The course is from a week to ten days. Recovery is the rule, though a cough may persist for weeks.

Treatment.—Avoid a cold, damp, drafty stable. The condition is aggravated by exposure to a wet cold atmosphere. Relief from the congestion and discomfort is secured by the use of medicated inhalations, such as half an ounce of creolin in a pail of hot water brought to boil just before use. In small stables a dish of medicated water may be kept steaming over a stove. Other useful inhalants are eucalyptol, turpentine, and composite tinct. of benzoin. An ice pack may give prompt relief, especially if there has been a recent traumatic injury of the mucosa. Some individuals receive greater benefit from a hot pack or poultice. Spirits of mustard and mild blisters are of doubtful value except in subacute and chronic forms. Sedatives and sedative expectorants are useful early in the disease for the relief of painful cough: For a horse, Dover's powder 1 to 2 drams (4-8 Gm.) three times daily, or apomorphine hydrochloride 1 grain (0.06 Gm.) hourly, or tartar emetic 2 to 4 drams (8-16 Gm.) in a pail of water night and morning, or atropine sulfate ¼ grain (0.015 Gm.).

℞ Ipecacuanhae ʒ i (4 Gm.)
Ant. et pot. tartratis ʒ i (4 Gm.)
Syrupi tolutani ℥ iv (120 cc.)
M. Sig.—One ounce (30 cc.) four times a day for a horse.

℞ F.E. belladonnae ℥ i (30 cc.)
Ant. et pot. tartratis ʒ iv (16 Gm.)
Syrupi O i (500 cc.)
M. Sig.—One ounce (30 cc.) every two hours for a horse or cow.

When the onset is intense, with high fever and pulse, tincture of aconite ½ dram (2 cc.) four times a day is beneficial. Constipation is best corrected by means of succulent food such as carrots or grass. Salines or oil may be given in small daily doses: Carlsbad salts 1 ounce (30 Gm.) in an electuary, or mineral oil 8 ounces (240 cc.). Careless administration of medicine, or the use of irritant drugs, easily causes choke and may induce a fatal pneumonia.

CHRONIC LARYNGITIS

Etiology.—Chronic laryngitis in domestic animals is infrequent. It occurs in a primary catarrhal form as a sporadic or epidemic affection in cows and horses. It may be secondary to pulmonary tuberculosis or other chronic lung affections. It may occur as a primary tuberculosis or actinobacillosis of the larynx, and it may follow an acute attack of laryngeal catarrh.

Symptoms.—Primary chronic laryngitis in cattle develops in stabled animals in the winter, and it may affect the greater part of a herd. The dominant symptom is a cough that develops gradually and persists until the cattle are turned to pasture; a few develop stenotic breathing, and there may be a loss of condition. Tubercles and other chronic growths in the larynx cause a gradually developing stenosis with inspiratory dyspnea. The prognosis is good in most primary forms, though improvement may not begin until warm weather. After an attack of strangles or pharyngitis there may be inspiratory laryngeal stertor which appears only when the horse is at work and that persists for weeks or months before it finally disappears. This symptom is apparently explained by Wirth[1] who has examined many cases of pharyngitis with the laryngoscope. These examinations revealed abscess formation with collateral edema affecting the lymph glands of the pharynx and the guttural pouches. The lymph glands of the guttural pouches lie above the arytenoid cartilages between the mucosa of the guttural pouches and the posterior wall of the pharynx. Delayed resorption of the edematous swellings is the probable cause of the inspiratory stertor. Persistent cough may be caused by chronic laryngeal

catarrh of unknown origin. This is most frequent in horses where it may lead to a suspicion of heaves while in cows tuberculosis may be suspected. Cough is easily induced by slight pressure over the larynx or upper tracheal rings. In a cow that came to autopsy after coughing for months a small red granular area was found on the mucosa of the larynx. Wirth reports that in chronic catarrh the mucosa is thickened and rough and somewhat dull, as seen through the laryngoscope.

Chronic laryngitis in feeder buck lambs in the early stages of intensive feeding of oats and barley in California has been attributed by Britton[2] to the wounding of the arytenoid cartilages by grain awns or other foreign objects.

Treatment.—Stimulant expectorants may be beneficial, yet they may have no apparent effect. To 500 cc. of the combination of camphor and ammonia given in coryza, one may add 1 ounce each (30 Gm.) of ammonium chloride and F. E. belladonna. Potassium iodide in doses of 1 dram (4 Gm.) daily for two or three weeks is sometimes effective in the horse; or one may give 30 Gm. of sodium iodide in 500 cc. of distilled water intravenously. An electuary of linseed meal, molasses, and 1 dram of belladonna extract to each tablespoonful of the mixture is prescribed by Dr. Law for persistent cough; a tablespoonful is smeared on the cheek twice daily. A light cantharides or mustard blister over the throat is sometimes useful. Removal from cold damp quarters may bring prompt relief. Benefit has been obtained from the following:

℞	Antimonii et potassii tartratis		
	Pulv. glycyrrhizae	aa ʒ x	(40 Gm.)
	Pulv. juniperi		
	Ammonii chloridi	aa ℥ iiss	(75 Gm.).

M. Sig.—A tablespoonful (15 Gm.) three times a day.

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EDEMA OF THE GLOTTIS

(Edematous Laryngitis; Edema of the Larynx)

Definition.—This is a serous infiltration with diffuse swelling of the mucosa and submucosa of the larynx that may occur independently, but more often is seen in association with general edema of the throat. It is not infrequent.

Etiology.—(*a*) As a form of anthrax in swine and blackleg in cat-

tle. (*b*) In purpura and malignant edema in equines. (*c*) In throat injuries—swallowed glass, metal, and wire, misuse of the probang, and abscess in the laryngeal wall. (*d*) After inhalants, as chloroform. (*e*) As an occasional form of urticaria in cattle. (*f*) In strangles. (*g*) As a symptom of anaphylaxis following the injection of a serum or bacterin, and from unknown causes in swine.

Symptoms.—In infection the development is sudden or gradual according to the intensity of the inflammation. After irritant inhalants the onset is sudden and the course rapid. Loud difficult inspiration is the leading symptom. When severe and unrelieved it soon leads to fatal suffocation. When caused by an infection, or when a part of an acute infectious disease, the prognosis is bad. Other affections of the larynx develop less rapidly.

In one instance in a horse that died after about forty-eight hours, the animal had been in the stable for a week and there was a possibility of a previous attack of strangles. Inspiratory stertor was marked, and there was a distinct fremitus to the touch over the throat. Slight pressure over the larynx caused paroxysms of coughing that threatened suffocation. Sweating was general, yet the appetite and condition were good.

In a cow, inspiratory dyspnea and slight swelling of the throat were first thought to be caused by laryngopharyngitis. Death from suffocation followed in a few hours. On the ventral border of the epiglottis there was an opening to a fistulous tract that passed backward as if a wire had penetrated the tissues, but no wire could be found. Multiple small abscesses surrounded the fistula. In another cow affected with inspiratory stertor, in which an extensive examination revealed no cause, autopsy disclosed multiple abscess formation in the depths of the laryngopharyngeal wall. A third cow presented bilateral swelling in the parotid region, inspiratory stertor and anxiety. Recovery followed the application of a compress of hot Epsom salts, one tablespoonful to a quart of water, followed by the application of a rubefacient liniment. Fatal edema of the glottis in swine has been attributed by Killham[1] to feeding of barley mixed with awns. Chronic ovine laryngitis with abscess formation and fatal edema of the larynx has been described in California by Cameron and Britton.[2] It was observed only in purebred yearling rams kept on full feed in the late fall and winter, and was attributed to ravenous eating of dry oats and barley containing a high percentage of awns. In advanced chronic atrophy of the laryngeal ligaments (roaring) in the horse, severe inspiratory dyspnea may lead one to suspect edema of the glottis.

Treatment.—For immediate relief insert a tracheotomy tube. Ad-

renalin in doses of 1 to 4 drams (4 to 16 cc.) may give prompt relief in urticaria and anaphylaxis. If the necessity is somewhat less, relief may follow inhalation of medicated steam, or the application of an ice pack or a hot compress to the throat. There are cases in which iodide of potassium 1 to 2 drams (4 to 8 Gm.) is beneficial. Sodium iodide (30 Gm. in 500 cc. of distilled water) intravenously has been recommended. Atropine sulfate may give relief. In specific infections, as anthrax or blackleg, the corresponding sera and antibiotics are useful when given early.

References

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CROUPOUS LARYNGITIS

Croup

Definition.—Croupous laryngitis is characterized by inflammatory swelling of the mucosa, a fibrinous exudate, and intense inspiratory dyspnea, usually it involves the trachea. It is a rare disease, affecting chiefly cattle between 6 months and a year old, and confined to certain localities. It is probably infectious, but no bacterial cause has been reported. Dampness seems to favor its development. The lesions are rarely confined to the larynx. Extensive fibrinous exudates are present in the nose and trachea, and the mucosa is severely inflamed. Pneumonia may also be found.

Symptoms.—The onset is sudden with a dry, painful, high-pitched and easily induced cough. Inspiration becomes difficult and stertorous. The mucosae are congested, the pulse and respiration fast, and the temperature high—105° F. Coughing may expel croupous shreds. The expression is anxious and suffocation threatens. Unless relief is prompt, coma and death soon follow. If the patient escapes early death from suffocation it may die later from toxemia. In an attack of croupous laryngitis in a 6-year-old cow in the latter part of June, the onset was sudden with high fever, extreme inspiratory stridor, and symptoms of choke. After two days fibrinous casts 4 inches long were expelled with violent coughing. The entire course was about a week, ending in recovery.

Diagnosis.—One should first determine whether the condition is an edema of the larynx, a croupous, or a diphtheritic inflammation. Grave laryngeal stenosis in calves suggests diphtheria; in swine, it may be

anthrax; in equines, it may be either anthrax or purpura.

Treatment.—For the relief of dyspnea, steam inhalation is most effective; this may be combined with the use of cold or hot packs over the throat. As a last resort, to prevent suffocation, one may insert a tracheotomy tube. The symptomatic treatment is the same as for edema of the glottis. Apomorphine is indicated.

NECROTIC LARYNGITIS

(Calf Diphtheria)

Necrotic laryngitis is a highly fatal disease of calves caused by *Actinomyces necrophorus* and characterized by deep necrosis. In Wyoming, the cases reported by Kingman[1] were chiefly in young stock weighing from 500 to 1000 pounds. It may be either sporadic or enzootic. It may occur as an independent slowly spreading epidemic in young calves in clean stables, but more often it occurs in association with necrotic stomatitis under filthy conditions. Injuries from rough feeds, thorns, thistles, barley beards, fox-tail beards, etc., are mentioned by Elder.[2] The lesions and etiology are the same as those of necrotic stomatitis in the young, but the laryngeal involvement is less frequent and more fatal. It is observed only occasionally in the Eastern States. While emphasis has been placed on the influence of filthy conditions and poor food, in New York the disease is more often met with under excellent sanitary conditions. It has not been observed here in pastured stock and it is most prevalent in the cold seasons, but may occur at any time. As described by Farquharson,[3] in the Rocky Mountain Region there is a high incidence in animals 6 to 15 months of age and occasionally it is seen in older animals; many cases are seen in feeder cattle and some in pastured animals up to 2 years of age; the disease is particularly prevalent through the winter and spring months and causes serious economic loss in both dairy and beef-cattle industries; there has been a gradual increase in the West from year to year.

Morbid Anatomy.—The larynx and epiglottis are covered with a purulent cheesy mass that entirely closes the opening. The necrotic mass extends deeply into the tissues. The lesions may be confined to the larynx or associated with necrotic stomatitis and pneumonia.

Symptoms.—The onset is sudden, with severe inspiratory dyspnea, a moist painful cough, and a temperature of about 105° F. The calf may take the usual amount of milk or refuse food entirely. Examination of the throat reveals grayish or yellowish necrotic deposits on the vocal cords and similar lesions may also be present on the cheeks and gums. In one or two days breathing becomes more difficult and weakness

develops. After a tracheotomy to relieve dyspnea, the animal appears to improve for a few days, but eventually the wound becomes infected, toxemia develops, and death follows. In a week or ten days a second calf may be attacked; it is not highly enzootic. The course, intensity, and complications are variable.

In a case described by Hastings[4] in a month-old calf showing high fever and a marked inspiratory dyspnea, it was found on autopsy that the necrosis involved the posterior part of the tongue, the soft palate, the pharynx and the larynx, as well as the trachea and a portion of the lung. In our clinic a 4-month-old calf that had been treated for pneumonia and necrotic stomatitis continued to snore for about five weeks like a horse with strangles. On autopsy the right vocal cord presented a small greenish white focus of caseated pus which proved to be the opening of a fistulous tract that almost completely encircled the first tracheal ring. The fistula was filled with fetid greenish white caseated pus. Both lungs were pneumonic.

Treatment.—While treatment of calves affected with necrotic laryngitis has in our experience usually been hopeless, the success of sulfapyridine, as described by Farquharson[5] in the treatment of feeder cattle, justifies its use. He reports that in the majority of cases recovery resulted from one treatment of 60 cc. of a 5 per cent solution of sulfapyridine sodium per 100 lbs. of body weight intravenously. The recommended daily dose is 45 grains (3 Gm.) per 100 lbs. of body weight. Care should be taken to avoid escape of the solution into the perivascular tissues. For the following two or three days one may prescribe 1 grain per pound of body weight orally, or repeat the intravenous injection in 24 hours. In one case in a 3-week-old calf treated in the ambulatory clinic, recovery followed the administration of sulfapyridine 15 grains (1 Gm.) every six hours orally. Treatment needs to be started at the onset of the attack.

In the treatment of an outbreak of diphtheria in nearly 3000 calves and feeder cattle following shipment in Montana, Hayes and Wright[6] reported excellent results from the administration of sulfamethazine. The drug was given orally with a balling gun, once daily for two days to each calf. Dosage varied from ¾ to 1½ grain per pound of body weight.

At the Iowa State College, aureomycin was highly effective in 14 cases of necrotic laryngitis in cows treated by Chivers.[7] *Corynebacterium pyogenes* was the predominant infection, and six had failed to respond to previous treatments with sulfonamides and penicillin. The dosage was 5 mg. per lb. body weight per vein at 24-hour intervals, sometimes increased as much as 500 per cent.

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ACUTE BRONCHIAL CATARRH

(Acute Tracheobronchitis)

Acute catarrhal inflammation of the bronchi is an affection of the large tubes—simple bronchitis. A similar affection of the smaller tubes was formerly termed capillary bronchitis, but this is now recognized as bronchopneumonia. Like coryza, bronchitis is common in all species in the cold wet months of fall and spring. Often it involves the larynx.

Etiology.—Basement stables, damp pens, concrete or stone walls, overcrowding, and low temperature are among the more frequent causes in calves and pigs. Poor ventilation or lack of ventilation also has an important influence. The ventilation of stables for young animals presents a special problem because the air they breathe is taken from near the floor, and it is difficult to bring fresh air to floors of small pens with tight walls. While bronchitis is an infectious disease that spreads by contact, its beginning often depends on poor housing conditions; after it is once established in the young, it becomes contagious. There are outbreaks, on the other hand, that are purely contagious and that spread among animals kept under ideal conditions. Little is known of the bacteriology, but in swine one may suspect the pneumotrophic virus of swine influenza, or the Pasteurella of swine plague. The active microorganisms are probably those which cause pneumonia, and in calves the question of whether a virus or the commonly found Pasteurella is the primary cause remains unanswered.

In other cases the housing may be excellent, but the pens are overcrowded. In all animals the disease may result from exposure to a cold draft when warm after exercise, from exposure at night after a warm day in the fall, from standing near doors that are frequently left open, and from transportation. In certain years it is epidemic in a mild

form in cows in the fall, and the addition of a purchased cow may be followed by an epidemic in the herd.

Acute bronchitis is a symptom of certain infectious diseases, as influenza, strangles, hemorrhagic septicemia, calf pneumonia, hog cholera and pneumotrophic infectious bronchitis in horses and cattle. It is also symptomatic of lungworm disease in calves and swine. In colts and swine, ascarid larvae in the lungs cause bronchitis and other pulmonary lesions.

Symptoms.—General symptoms are often limited to dullness and anorexia. In cows there may be a loss in condition and a decrease in milk. In colts the onset may be sudden and marked by congestion of the mucosae, an increase in the pulse and respirations, and a temperature of 103.0°-105.0° F. Cough is frequent and easily induced by slight pressure on any part of the trachea. Cows may lower the head, open the mouth, and protrude the tongue as if choked. Vesicular murmur is increased and a few coarse râles or loud bronchial sounds may be heard. Percussion of the chest is negative. The course is short, three days to a week, and the prognosis is good. In the enzootic form in cows the first and leading symptom is a dry suppressed cough; in about a week another animal begins to cough, and finally the entire herd may develop various degrees of bronchitis, ranging from an occasional cough to poor condition and decrease in the milk flow. In a few the disease becomes chronic. In calves the chief danger is the extension of the process downward. After all other symptoms have subsided and the calf is otherwise apparently well, there may remain an increased frequency and intensity of breathing to suggest an involvement of the lungs.

Diagnosis.—One must differentiate between bronchial catarrh and pneumonia. When bronchial catarrh develops in damp cold quarters it usually leads to pneumonia. The latter disease is marked by a rise in the pulse, respiration, and temperature, and by dullness and anorexia. These alone, in an acute bronchial disease, justify a diagnosis of pneumonia. When râles or marked changes in the vesicular murmur are present, the diagnosis becomes positive. Calf pneumonia in a mild form is not easily differentiated from acute bronchial catarrh. It is a common error to mistake pneumonia for bronchitis. Lungworm disease in calves and pigs may be mistaken for either acute or chronic bronchitis. Enzootics of bronchitis following the introduction of new cows are possibly mild types of hemorrhagic septicemia.

Treatment.—Complete rest in a warm dry stable is of first importance. In suitable weather remove to a sheltered cover open on one side. Cough may be relieved by ammonium chloride and ammonium carbonate, as recommended for coryza. Medicated steam inhalations are

beneficial. If the cough is dry and irritating, give to a horse Dover's powder in doses of 1 to 2 drams (4-8 Gm.) three times a day. Of the sedative expectorants, pilocarpine hydrochloride 8 grains (0.5 Gm.) three to four times a day is among the best. Other sedative expectorants are

℞ Ipecacuanhae	
Anti. et potassii tart.	aa gr. xxx (2 Gm.)
Syrupi	℥ iv (120 cc.).

M. Sig.—One ounce (30 cc.) four times a day for a horse or cow.

℞ F. E. Belladonnae	℥ i (30 cc.)
Anti. et potassii tart.	ʒ iv (16 Gm.)
Syrupi	O i (500 cc.).

M. Sig.—One ounce (30 cc.) every two hours for a horse or cow.

As in other respiratory affections, chemotherapy may be given, such as procaine penicillin, 1,500,000 to 3,000,000 units for large animals daily; sulfamethazine, 1 grain per lb. of body weight orally per day for two or three days; terramycin, 1 mg. per lb body weight per day administered intravenously at one time.

Whenever possible the first one to be attacked should be removed to other quarters, especially if there is a fever. When animals are removed because of sickness, new additions to maintain the original number should not be made.

CHRONIC BRONCHITIS

Etiology.—Primary chronic bronchitis may follow a severe acute attack, but more often it is chronic from the beginning. Usually it is secondary to chronic diseases of the lungs, as tuberculosis, glanders, lungworm disease, heaves, and pulmonary abscess in cows. In cows chronic bronchitis is suggestive of tuberculosis, yet it may occur in this species as an independent nonspecific infection.

Morbid Anatomy.—The bronchial mucous membrane is swollen, red, and covered with mucus. Small pneumonic foci are usually present in the lungs. In the horse the lungs are emphysematous, as in heaves.

Symptoms.—The symptoms of *secondary* chronic bronchitis depend on the nature and intensity of the primary disease. Usually one finds a lowered general condition, a cough, and abnormal respiratory sounds. *Primary* chronic bronchitis begins with a short dry cough. After a time this becomes moist and low; it is never painful, and is easily induced by pressure over any part of the trachea. The breathing is expiratory in type—"heavy." Percussion is negative. Loud bubbling musical râles may be local or extensive, and there may be bronchial fremitus, espe-

cially in cows. Finally there is a loss in weight. Exposure to storm aggravates the condition. Under favorable circumstances recovery takes place in a few weeks or months. Infrequently the disease spreads extensively in the lungs. Emaciation now appears; the cough is low and moist, and bubbling râles are distinct. In well-marked types a purulent exudate is abundant; this appears at the nostrils in the horse, but is swallowed by cows.

Diagnosis.—It is important to exclude bronchopneumonia and heaves. In cows pulmonary tuberculosis should be considered with respect to exposure to infected animals, reaction to tuberculin, and guinea-pig inoculation with bronchial secretions. In all well-marked chronic bronchitis it may be taken for granted that the lungs are affected, either with pulmonary emphysema, or foci of pneumonia, or both.

Treatment.—A summer at pasture is the best remedy. Stimulant expectorants, bitter tonics, and intensive feeding are useful through the winter. As in all other respiratory affections, the subject should be protected against exposure to atmospheric extremes and fatigue. The following expectorants are useful: expectorant of ammonium carbonate and ammonium chloride and camphor, as prescribed for colds.

℞ Antimonii et potassii tartratis	℥ i	(30 Gm.).
Ammonii chloridi		
Pulv. fruct. juniperi	aa ℥ iii	(90 Gm.).

M. Sig.—Tablespoonful (15 Gm.) in the feed three times a day.

℞ Calcii hydroxidi		
Sal carolini factitii	aa ℥ xvi	(500 Gm.)
Arseni trioxidi	ʒ x	(40 Gm.)
Lobeliae	℥ iv	(120 Gm.)
Belladonnae	℥ i	(30 Gm.).

M. Sig.—Tablespoonful (15 Gm.) thrice daily for a horse.

ACTIVE CONGESTION OF THE LUNGS

English and American medical authors, in general, deny the existence of an independent primary active congestion of the lungs, regarding it as a symptomatic affection. Yet the veterinarian often meets with a condition in the early stages of pneumonia in cattle and horses that is diagnosed and treated as active congestion.

Active pulmonary congestion in cattle and sheep has been reported in Jugoslavia by Kucel (Abs. Vet. Bull., 1937, 7, 627). The disease was observed in the spring when the animals were first turned to pasture and appeared to be related to sudden changes of weather. The onset was sudden with tremors, dyspnea, fever, and indigestion. Pulmonary edema which resembled anthrax was found to be the chief lesion.

Etiology.—(*a*) Exposure to storm and fatigue. The animals have been subjected to cold rains, cold nights, or transportation in winter; it is a disease of the cold seasons. (*b*) The first stage of pneumonia (infection). (*c*) Inhalation of hot air or irritant gases is an infrequent cause. (*d*) Extreme overexertion, as in hunters and racers, induces a special form secondary to acute heart failure.

Morbid Anatomy.—When death occurs the changes are those found in the initial stage of lobar pneumonia. The lungs are distended, dark red, or black in color, and contain enormous quantities of blood. The cut surface exudes dark blood or serum. Pieces dropped in water may either sink or float below the surface. Under the microscope one finds a marked engorgement of the capillaries, and a slight exudation of serum, fibrin, or cells into the alveoli.

Symptoms.—These vary according to the degree of congestion and the species. In horses the attack is usually at night, following a hard day's work. There is a history of dullness, poor appetite, sometimes restlessness, even colic, and fast breathing. Pneumonia is suspected. One finds depression, congested mucosae, cool extremities, and possibly chills. The pulse is 75 to 100, the breathing 25 to 30, and the temperature 104°-106° F. Breathing is intense and often abdominal. A slight serous exudate is present at the nostrils. Cough is occasional, low, and with few exceptions is easily induced. The vesicular murmur is harsh and a few crepitant râles may be heard. On percussion the tone is either normal or tympanitic. When congestion does not go on to pneumonia improvement takes place in a few hours.

In cows the initial symptoms are dyspnea and a diminished milk flow. The pulse and breathing may be fast, and the temperature normal or high. There is an expiratory grunt, a short harsh cough, and marked dyspnea—head down, mouth open, frothing as in choke, and the froth may be blood-stained for in intense congestion there is always some pulmonary edema. Sibilant râles are not infrequent. In mild forms the symptoms are similar but less intense.

The usual form, due to exposure, responds promptly to rest and treatment. When the affected subjects are worked they soon collapse, and death from extensive congestion may follow in a few hours. A highly fatal type occurs after long exposure in transportation. Inhalation congestion leads to gangrenous pneumonia.

Diagnosis.—Only by the brief course can it be distinguished from pneumonia. The early stages of acute febrile or painful disease, as laminitis, anthrax, and strangles, may resemble active congestion in the rapid harsh breathing and the fever; but these alone are not distinguishing symptoms of any disease.

Treatment.—Rest in a protected dry place is of chief importance in the handling of exposure cases. An expectorant of ammonia and camphor, as advised for coryza, one ounce every two to four hours, is useful. In marked dullness one may prescribe strychnine sulfate (gr. i) thrice daily. When the patient is restless and in pain, a mustard paste or spirits of mustard over the chest may bring prompt relief. In the presence of marked cyanosis and dyspnea, as after extreme exertion, removal of from four to six quarts of blood from the jugular vein affords relief to the lungs and heart; but this is of little value except in the beginning, when the dyspnea is marked and the mucosae are highly congested. Other respiratory sedatives are atropine sulfate ¼ grain (0.015 Gm.) with lobeline sulfate ½ grain (0.03 Gm.). Vascular tension may be relieved by spirit of glonoin ½ to 1 dram (2-4 cc.) or glonoin ¼ to ½ grain (0.015-0.03 Gm.) every hour until the breathing and pulse are improved. Sulfonamides are indicated.

PASSIVE CONGESTION OF THE LUNGS

This is a secondary condition caused by a weak right heart. It results chiefly from: *(a)* Chronic dilatation of the heart, especially in the horse. The subject is usually aged and suffers from nosebleed and collapse when worked. *(b)* Heart failure in acute general disease. *(c)* Hypostasis, determined largely by the influence of gravity in animals that are unable to rise. In passive congestion associated with acute disease the condition is recognized only on autopsy.

ACUTE PULMONARY EDEMA

Primary acute pulmonary edema is characterized by a sudden attack of dyspnea due to extensive transudation of serum into the lung tissue, including the alveoli, bronchioles and bronchi. The cause is unknown. As an independent clinical disease it is infrequent in domestic animals. Collateral edema, limited to the area of the affected part, occurs in various pathological lesions in the lungs. Terminal edema, due to hypostasis, or circulatory weakness, is common. And pulmonary edema as a complication of anthrax, malignant edema, and purpura is not infrequent. In all forms of pulmonary congestion there is some edema. But in primary acute pulmonary edema there is no evidence that it is an extreme stage of acute congestion of the lungs or that it is secondary to other conditions or that it is associated with other evidence of circulatory failure. In animals that die of anaphylaxis following the administration of serum or bacterins, extensive pulmonary edema is the chief lesion, a suggestion that primary acute pulmonary edema is caused by some obscure toxic influence.

In medical texts the chief causes of pulmonary edema are given as *(a)* heart failure, or increased permeability of the capillary walls as in shock; *(b)* infection, either as a complication of infectious diseases, or the result of a toxic effect on the centers controlling the vasomotor system of the lungs; and *(c)* toxic poisoning from morphine, adrenalin, iodine, or alcohol, and inhalation of irritating gas.

Symptoms.—The following record was made on one case of acute primary pulmonary edema: This was in a 5-year-old nonpregnant cow in the ninth month of lactation in a large dairy herd. The morning of May 23 she ate little and gave little milk. In the afternoon she was very ill, dyspneic, and shivering. On being moved to another stall she discharged at least a gallon of clear serum from the mouth and nose and shortly thereafter she died. On post mortem examination a large amount of clear serum was found in the chest cavity. There was an extensive diffuse edema of the lung; it was heavy and pitted on pressure. When incised, an abundance of clear serum flowed through the channel made by the knife. There was also considerable emphysema. The bladder contained urine stained nearly black with hemoglobin. No other lesions were found. Various organisms were found on bacteriological examination of the lungs, among them a few Pasteurella.

A second case was observed by Dr. Fincher in the ambulatory clinic in a 7-year-old dairy cow twelve hours after intradermic injection with tuberculin. The symptoms developed rapidly. There were fast breathing, expiratory grunt, increased vesicular murmur and râles. The temperature was normal. Pulmonary edema and emphysema were found on autopsy; it was believed that the emphysema was secondary.

Five additional cases have been observed, four of which were young bulls from 8 months to 2 years of age. The onset has been sudden with labored fast breathing and expiratory dyspnea with fibrinous exudate or blood-tinged foam at the nostrils. Auscultation has shown increased vesicular murmur and moist râles. The temperature has been normal or as high as 104° F. The respirations have been from 60 to 80, and the pulse from 80 to 100. Hemoglobinuria has usually been present. The course has been two to twelve hours ending in death, or two to three days with recovery. In milder forms, "summer snuffles," and acute indigestion have been suspected at the onset of the attack. On *autopsy* there has been extensive edema of the lungs and the bladder has contained urine stained nearly black with hemoglobin. In one there was a hemorrhagic tracheitis. Pulmonary edema of swine, associated with pneumonia, has been described by Murray and Biester.[1]

Treatment.—In anaphylaxis adrenalin (5 to 10 cc.) is probably the most effective remedy. This is often combined with atropine ¼ grain

(.015 Gm.), but the value of atropine in edema is questionable. The use of antihistamine compounds (pyribenzamine, benadryl) may be of distinct benefit.

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HEMOPTYSIS

(Pulmonary Hemorrhage)

Etiology.—*(a)* Hypertrophic dilatation of the heart is the most frequent cause in the horse. *(b)* Multiple pulmonary abscess formation is the most frequent cause in the cow. *(c)* Erosion of a blood vessel occurs infrequently in cows affected with pulmonary tuberculosis; and free hemorrhage may occur in cows affected with a terminal pneumonia in septicemia. *(d)* Rupture of the pulmonary vein has been observed in the horse. *(e)* In an aged horse affected with marked hemoptysis, thrombosis of the hepatic artery, dilatation of the right heart, and a pulmonary infarct were found. *(f)* Capillary hemorrhage is rather frequent in the congestive stage of lobar pneumonia in equines. Intense active congestion of the lungs from any cause may lead to edema with hemoptysis. Fatal hemoptysis in a cow caused by occlusion of the left jugular vein by an abscess has been described by Fincher.[1] In one instance, on arrival of a carload of horses one animal was found dead from extensive pulmonary hemorrhage of unknown cause. Pulmonary hemorrhage in swine following vaccination against hog cholera has been reported by Kinsley.[2]

Morbid Anatomy.—Hemoptysis is immediately fatal only when an erosion, or an aneurism, leads to rupture of a vessel. A break through the pulmonary vein may extend through the visceral pleura and discharge much blood into the chest cavity where it forms a large clot; blood in also found in the bronchi and air cells.

Symptoms.—Usually there is a history of "nosebleed." In erosion of a vessel, bright-red foamy blood discharges freely from both nostrils. If the hemorrhage is only moderate, clots of blood form in the nostrils and these may be expelled by coughing or sneezing or they may remain for hours. Following an attack of indigestion in a cow, fluid and foam and blood came from both the nostrils and the mouth; bloody foam at the mouth and nostrils is symptomatic of intense active congestion of the lungs. In a fatal case of traumatic gastritis and multiple abscess of the liver in a cow, long clots of blood and mucus hung from the nostrils. In another fatal case of traumatic gastritis, terminating in pyosep-

ticemia, there was free hemorrhage from the lungs. In the case of occlusion of the left jugular vein there was repeated hemorrhage mixed with foam from both nostrils over a period of a month. Finally, continuous coughing and extreme hemorrhage developed when the cow was being led and death occurred suddenly after a loss of about 10 liters of blood. On examination after the hemorrhage has ceased, one will find râles and other signs of the chronic lung disorder. In the horse chronic hypertrophy of the heart may lead to bleeding of the nose in the form of clots when the animal is stabled, or in the form of blood mixed with air when worked. The underlying heart disease is revealed by the palpitating irregular heart beat and the poor quality of the pulse.

Rupture of the pulmonary vein causes weakness, collapse, and convulsions, that lead to death in a few minutes or a few hours; blood may not appear at the nose in the final attack. The associated restlessness is suggestive of colic; but in hemorrhage the mucosae are pale, while in severe colic they are red. The extreme dyspnea and delirium following rupture of a large pulmonary vessel have led to a mistaken diagnosis of suffocation from foreign body in the trachea.

Capillary hemorrhage, hemoptysis in the narrow sense, is recognized in the horse by the presence of a small amount of dried blood at the margin of the nostril. This may be black, brown, or yellowish in color, and is pathognomonic of pneumonia. In chronic affections hemoptysis is a symptom of an incurable disease of the heart or lungs. In acute pneumonia in cows free bleeding from the nose is a fatal sign. Bleeding that develops during exercise usually stops in a few minutes after the animal comes to rest. In chronic dilatation of the heart, clots appear at the nostril from time to time, but usually the bleeding is not marked.

Treatment.—Aside from complete rest the effect of treatment is in doubt. The handling of the case depends on the underlying cause. Because of the variable nature of the underlying causes of hemoptysis in large animals, treatment is not commonly effective. Agents for the treatment of epistaxis may be useful: atropine sulfate, pituitrin, adrenalin, sodium citrate, or blood transfusion.

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HEAVES

(Broken Wind; Pulmonary Emphysema; Asthma)

Definition.—Heaves is a chronic specific disease of equines characterized by expiratory dyspnea, a wheezy cough, and intestinal fermentation, finally leading to emphysema of the lungs. By some it is regarded as a functional respiratory affection of dietetic origin—a neurosis. Hutyra and Marek[1] write that the emphysema is caused by prolonged tension of the lung tissue, as in chronic bronchitis, chronic cough, or extreme exertion.

Etiology.—The essential cause is unknown. It follows excessive feeding of dusty hay and clover to stabled animals. The malady does not develop in pastured horses, or in those whose food is limited to straw and barley, to natural hay fed sparingly, or to cornfodder and bran. According to Williams,[2] it does not occur in horses fed on timothy and clover that have been raised under irrigation. Dr. Law[3] writes that it is unknown among the horses of the Arabs; that it is rare in Spain, Portugal, and certain parts of France and England; that years ago it was virtually unknown in Michigan and adjacent states, and that California long retained the reputation of having no broken-winded horses. With the introduction of timothy hay, red clover, alfalfa, and other cultivated grasses, the disease has become prevalent in most parts of the United States. It is more frequent where the climate is unfavorable to the curing of hay, and where farm horses spend weeks at a time in the barn with hay constantly before them. Prolonged exhaustive work with the associated heavy breathing helps to bring on the attack. Prolonged dyspnea from any cause leads to pulmonary emphysema, but when the dyspnea is relieved the lungs return to normal.

According to Hug,[4] who examined 38 affected lungs, chronic dyspnea or heaves may be caused by any one of several pulmonary lesions: most often it is chronic bronchitis, then follow bronchitis and pleuritis with fibrosis, and less frequently tumor formation. In these conditions, emphysema is attributed chiefly to persistent cough which causes high intra-alveolar pressure. The conclusions drawn by Hug represent the general view in Continental Europe. He reports a tendency to bronchial catarrh in the acclimatization diseases of horses imported into Switzerland, in horses kept in warm unhygienic cow stables, and in those fed upon dusty hay and straw. In Switzerland 25 per cent of indemnities for horse insurance is paid for losses caused by heaves, and these losses increase following a season when the harvesting of hay is difficult. Nielsen[5] in Norway has observed that dusty and mouldy hay play a

more important part in the cause of heaves than is generally believed. According to his view dust and fungi cause bronchitis to which emphysema is secondary. Whichever view one accepts as the primary cause of pulmonary emphysema: dietetic neurosis, or chronic bronchitis, there is no difference of opinion concerning the influence of damaged hay or clover. It is apparent that chronic bronchitis in any species, including man, may cause pulmonary emphysema.

With the exception of heaves, chronic bronchitis is not common in horses. The usual form is acute in association with one of the infectious diseases of the respiratory system, such as influenza; and these affections occur chiefly in the young. In horses destroyed because of advanced pulmonary emphysema, the presence of chronic bronchitis on autopsy does not prove it to be the primary cause of the disease. Because of the distinctive characteristics of heaves, it is probable that both the emphysema and the bronchitis are due to a common cause the essential nature of which is unknown.

The view that the disease may be inherited is not generally accepted. That such a condition is possible may be inferred from knowledge on hypertrophic emphysema in man where an hereditary influence has been proved and where a nutritive change in the air cells is widely accepted as primary.

It is now believed that work requiring prolonged severe exertion does not predispose to an attack. Yet the symptoms may be made worse rapidly when a green horse affected with heaves is placed at heavy work, and in some instances this influence is the apparent cause of the disease. In the terms of the farmer, "the horse broke its wind on the plow."

Heaves rarely affects a horse before the fifth year and its frequency increases with age. In our clinic nearly all have been at least eight years old. It is most frequent in draft animals.

Morbid Anatomy.—Alveolar and interstitial emphysema are the main visible changes. On removal of the chest wall the lungs are found so greatly distended that they carry the imprints of the ribs. The organs have lost their elasticity and do not collapse. The pleura is pale and beneath it are air vesicles. The margins of the lungs are thickened and the tissue pits on pressure. Chronic bronchitis is always present. The alveolar walls atrophy, the capillary network disappears, and the adjacent alveoli unite to form a single enlarged air cell. The thickness of the alveolar walls is greatly reduced. Owing to the diminished vascularity of the lungs, the right side of the heart is hypertrophied.

Symptoms.—As a rule heaves is insidious in development, but some-

times it develops suddenly and in a severe form during hard work in the spring. There is a history of one or more of the following symptoms: *(a)* cough and slight nasal discharge after drinking; *(b)* a cold followed by wheezy night and morning cough; *(c)* coughing spells all winter and easily fatigued; *(d)* rattling in the head. The breathing shows a double expiratory lift of the abdominal muscles, which compensates for the loss of elasticity in the lung, and the compensatory lift is greatly increased by exercise. At the nostrils one often finds a slight dilation and a slight bilateral serous nasal discharge. The cough is prolonged, low, wheezy, moist, and easily induced; it is pathognomonic of the disease. At first it may be harsh and paroxysmal. The lungs give wheezing, sibilant, or crepitant râles, or an increase in the vesicular murmur. In advanced cases there is an increased area of resonance on percussion. Although a ravenous eater, the victim is often unthrifty. Because of obstruction to circulation in the lungs the conjunctival mucosae are congested. The condition is aggravated by humid atmosphere, heavy work, overfeeding, colds, and exposure to storm. It is incurable, and under usual conditions is progressive until the animal becomes worthless. Recoveries have been reported; but it is probable that these were from a primary bronchitis, a disease that closely resembles the early stages of heaves. The following digestive symptoms are often met with: ravenous appetite, large abdomen, excessive bowel fermentation, and frequent passage of flatus.

Treatment.—Give regular light work and protect against storm. Limitation of roughage to straw or cornstalks is seldom practical. Feed lightly on clean timothy hay free from clover and sprinkled with lime water. A season at pasture is beneficial. In conjunction with improved care and feeding the following prescription is useful.

℞ Artificial Carlsbad salts	
Calcium hydroxide	aa ℥ xvi (500 Gm.)
Arsenic trioxide	ʒ x (40 Gm.)
Lobelia	℥ iv (120 Gm.)
Belladonna	ʒ i-iv (4 to 16 Gm.).

Mix. Tablespoonful (15 Gm.) thrice daily in the feed.

For many years Fowler's solution of arsenic has been used to alleviate the symptoms. It may be combined with respiratory sedatives as follows:

℞ F. E. lobelia	
F. E. belladonna	aa ℥ i (30 cc.)
Fowler's solution q.s.	O i (500 cc.).

Mix. One tablespoonful (15 cc.) twice daily in two or three quarts of drinking water.

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PULMONARY EMPHYSEMA OF COWS

Interstitial and alveolar emphysema commonly develop whenever there is a persistent severe dyspnea, regardless of the species or the cause. In cows affected with pneumonia, extensive interstitial emphysema is common; it has been found in an extreme form in cows suspected of having hemorrhagic septicemia (see page 449), and in those known to have died of lead poisoning. Old cows that have been condemned on physical examination because of râles and suspected pulmonary tuberculosis often have shown only emphysema of the lungs on postmortem examination. It has been found in pulmonary abscess, in edema of the lungs, following perforation of the lung by a wire in traumatic gastritis, and in lungworm disease of calves.

Symptoms.—In association with multiple lung abscess and pleuritis, there may be a sudden attack of dyspnea accompanied by mouth breathing and the presence of white foam at the mouth and nostrils. Coarse, crepitant râles are widely heard over either side of the chest, and subcutaneous emphysema may finally appear. The course and termination depend on the nature of the primary disease. As an apparently independent disease of unknown cause it is observed occasionally in cows where there may be a history of sickness for several days with symptoms suggestive of traumatic gastritis. Towards the end the breathing is rapid and grunting, the animal may be barely able to stand, the eyes are sunken, drooling is prominent, and subcutaneous emphysema is extensive. The pulse may be 100 or more and usually there is a fever.

Treatment.—Adrenalin chloride (2 to 3 cc. injected subcutaneously) may afford prompt but transient relief. Relief may also be obtained from the combination of salines and sedatives prescribed for heaves.

BRONCHOPNEUMONIA

(Inflammation of the Lungs; Catarrhal Pneumonia; Capillary Bronchitis)

Inflammation of the lungs is difficult to classify and describe. This is expressed by Fröhner[1] in the statement that, "according to the character, the location and extent, the cause and the course, no other organ

in the body presents so many forms of inflammation"; and by Hare,[2] in discussing bronchopneumonia, that "the affection is so varied in cause and pathology as to lead to the suggestion that it be called a lesion rather than a disease."

In text-book descriptions, both medical and veterinary writers apply the terms *lobar pneumonia* and *bronchopneumonia* to the chief clinical forms. In human medicine these are described by Osler[3] in the section on specific diseases. In veterinary medicine lobar pneumonia is usually described under both contagious equine pneumonia and ordinary non-contagious equine pneumonia; although some believe that no distinction between the two exists. So-called lobar pneumonia in the horse is unlike lobar pneumonia in man, both in pathology and bacteriology. Since bronchopneumonia, with rare exceptions, is the type in domestic animals, the term lobar pneumonia is used in this text only to indicate an extensive diffuse involvement of one or both lungs.

Definition.—Bronchopneumonia is usually an extension of a tracheobronchitis that involves a few or many foci or lobules in the lungs. It is the usual type in domestic animals. Consolidation may be gradual and slight, involving only a few lobules (lobular); or it may be rapid, extensive, and continuous (lobar). It may at first be gradual and slight and after a time suddenly become diffuse. Bronchopneumonia is either primary or secondary. The secondary form is by far the more common, and when pneumonia is recognized one should learn whether it belongs to this type. It occurs chiefly in the fall and winter. All species are attacked, but it is most prevalent in cattle.

Etiology.—(A) PRIMARY ACUTE BRONCHOPNEUMONIA.—(1) *Chilling.*—There has been much discussion on the relation of exposure to acute respiratory disease. Many look upon chilling as secondary in importance to infection, as merely lowering the resistance of the bronchial and pulmonary tissues. Both operate together, and in individual cases either may dominate. While infection occupies first place as the cause of pneumonia, the effect of chilling is often so obvious, especially in the young, that it may require chief consideration both in prevention and treatment. Pneumonia caused by exposure tends to be sporadic, but the bacteria normally present in the bronchi may increase in virulence and be carried to other animals. Basement stables with walls of stone or cement favor the disease. Calves readily contract pneumonia when exposed directly to drafts that enter windows or other air intakes, but may escape respiratory diseases entirely when kept with nurse cows in well-bedded and tightly constructed sheds open on one side. Colts contract pneumonia when heated in the warm hours of the day and then chilled in cold stables at night. Pigs that huddle to keep warm

are chilled when they scatter. Pneumonia often develops in animals that have been heated and then rapidly chilled, have stood in cold rains, been exposed in transportation, or left in the pasture in the first cool nights in the fall.

(2) *Infection.*—The more prevalent contagious forms of bronchopneumonia are described in the section on specific infectious diseases; they include contagious equine pneumonia, stockyards pneumonia in cattle (hemorrhagic septicemia), swine influenza, and lung plague. In sporadic pneumonia in cattle, sheep, and swine, it is probable that often the infective agent is identical with that causing enzootic pneumonia. But the nature of the infective agent is not generally known. A pneumotrophic virus has been demonstrated to be the cause of swine influenza, equine influenza, possibly equine contagious pneumonia, enzootic equine laryngotracheitis (bronchitis), a similar form of enzootic bronchitis in Germany, and pneumonia in calves.

Whether or not the *Pasteurella* group is the primary cause of pneumonia in animals is a controversial subject. But until some other organism can be found its presence will be regarded by clinicians as at least suspicious. In pneumonia in swine one needs to consider the possibility of hog cholera, and swine influenza.

Enzootics of pneumonia due to undetermined infection have been met with in various parts of the world. A contagious form in cattle in northern Switzerland in the fall and winter has been described by Wyssman;[4] infection was carried by direct and indirect contact. An affection in cattle in Holland termed autumn lung disease is a bronchopneumonia from which van der Ween[5] isolated pyogenic bacteria, mostly *Corynebacterium pyogenes;* it was not prevented by removal to fresh pastures. The associated catarrhal bronchitis led to emphysema, and intestinal catarrh was common. *Pseudomonas pyocyaneus* was regarded by Birch and Benner[6] as a cause of fatal pneumonia in swine. In Montana and adjacent states a fatal bronchopneumonia in sheep has been recognized since 1915—Hadleigh Marsh.[7] The annual loss in affected herds averages from 2 to 10 per cent. Dr. Marsh believes it to be infectious, and that the primary bacteriological factor is a diphtheroid bacillus closely resembling *Corynebacterium pyogenes.* It has been reported only from range sheep exposed to large amounts of dust during shearing time and when trailed to and from summer ranges. Extensive studies by Creech and Gochenour[8] failed to disclose any bacterial cause for the disease. From India and East Africa a contagious pleuropneumonia affecting sheep and goats has also been reported—Poulton.[9] See enzootic pneumonia of sheep.

(B) Secondary Bronchopneumonia.—As a *sequence of infectious*

diseases, this type is by far the most common lung disease. In cows it is associated with mastitis, traumatic gastritis, metritis, and other acute or chronic infections. In metritis the pneumonic onset may even mask the metritis until it is revealed by autopsy. It may be secondary to intestinal catarrh in the young, and to equine influenza. Pneumonia in swine is nearly always secondary.

(C) Gangrene of the Lungs is a putrefaction of the lungs following necrosis. It is infrequent except in the horse. Usually it is a part of inhalation pneumonia caused by drenching; it is also a complication in severe types of contagious equine pneumonia; and it is one of the chief lesions of chronic lead poisoning in equines—vagus pneumonia. In a case of traumatic gastritis in a 5-year-old cow a fistulous tract penetrated the endocardium of the right side of the heart, purulent material entered the blood stream, and multiple gangrenous areas from 5 to 7 cm. in diameter formed in the lungs.

(D) Vagus Pneumonia has been produced experimentally in the dog by separation of the pneumogastric nerve. It occurs in horses and other animals from the paralyzing effects of chronic lead poisoning—Haring and Meyer.[10] The horse also suffers occasionally from a paralysis of the throat of unknown origin that may end in deglutition pneumonia.

(E) Lungworm Disease in swine, sheep, and calves, and ascarid larvae in pigs and foals, cause pulmonary inflammations that are distinctive of each.

Morbid Anatomy.—In acute bronchopneumonia the consolidation is located in the anterior dependent portions of one or both lungs. The pleural surface presents reddish or grayish red slightly raised areas of hepatization. When the inflammation has spread slowly, these may be isolated patches surrounded by normal lung (lobular pneumonia); when it has spread rapidly, they may involve a large part of one or more lobes (lobar, sometimes termed pseudolobar pneumonia). Less frequently, the changes are in the bronchi and bronchioles with no definite consolidation, yet the alveoli contain inflammatory products. Other reddish areas, slightly sunken, are parts from which the air has escaped (atelectasis). In intense inflammations one may find serum in the thoracic cavity and fibrin over the pleura. Hemorrhage on the pleura is common. When dyspnea has preceded death, the dorsal and posterior parts of the lungs often present extensive emphysema, especially in bovines. In cattle and swine the pleural surface often is marked by radiating bands of thickened interlobular connective tissue. On *section* the affected lung is either red or gray, a combination of both, or a red surface is sprinkled with small grayish areas. The bronchial mucous membranes are inflamed. The bronchioles contain grayish or yellowish

pus, and the adjacent peribronchial tissue is consolidated. Scattered areas of consolidation represent groups of lobules or parts of lobes. Hemorrhage, emphysema, and interlobular thickening are frequent. In the more severe types, and in those caused by certain kinds of infection, as *Corynebacterium pyogenes*, abscess formation and necrosis develop. Lung abscess is common in secondary pneumonia in bovines, in vagus pneumonia in equines, and whenever the course of any pneumonia is prolonged to two or three weeks. In chronic pneumonia there may be swelling and abscess formation in the mediastinal lymph glands, as well as laryngitis and rhinitis with diphtheritic patches on the turbinated bones. On cross section of a lobule the inflammatory changes immediately surrounding the bronchioles and smaller air passages are less recent than those farther away. From the primary area of attack the inflammation spreads along both the transverse and longitudinal axes of the air passages.

Suppuration, necrosis, and gangrene, are prominent lesions in inhalation pneumonia.

Symptoms.—An increase in the rate of respiration, and cough, are the usual early symptoms. These may be preceded by fever, depression, anorexia, and diminished milk flow. In secondary pneumonia the lung affection may, in certain cases, be recognized only on autopsy. Usually the nature of the primary disease warns of the impending danger—severe metritis or mastitis. Occasionally, secondary pneumonia is the dominant feature from the onset, even masking the primary infection. On recognizing pneumonia, one should determine whether it is primary or secondary.

The onset of the primary form may be abrupt, often in a damp stable, or following transportation, or after exposure to the sick, and usually in the cool season of the year. The general symptoms are marked. The conjunctival mucosa is usually congested in equines, and it may be congested in cattle. The pulse ranges from 60-100, the respirations are 40-90, and the temperature is from 103°-106° F. In cattle and swine affected with secondary pneumonia the temperature may be normal. The breathing is labored and cattle often give an expiratory grunt. Open-mouth breathing, with extension of the tongue, retraction of the commissures of the lips, and frothing, seen in cattle, indicate grave involvement of the lungs. In severe dyspnea, emphysema of the lungs may lead to rupture of the lung tissue and an accumulation of air beneath the skin: survival of animals thus affected is rare. The *nasal discharge* varies. In equines it is usually slight and dries at the nostrils; if the larger bronchi are affected, it may be abundant. In cattle a mucous nasal discharge is common, and blood at the nostrils indicates

either pulmonary abscess, or intense active congestion with pulmonary edema. In swine a secondary suppurative pneumonia caused by infection with *Corynebacterium pyogenes* may give a purulent nasal discharge. *Lung gangrene* in equines gives to the nasal discharge and breath a sweetish highly fetid odor. *Cough* is usually present and easily induced, though in metastatic forms, as metritis, it may be absent and uninduced.

Auscultation gives anything from absence of sound to increased vesicular murmur and a wide variety of râles. In extensive pleuropneumonia of traumatic gastritis there may be harsh, wheezing, dry râles. In pneumonia caused by drenching one is apt to hear bubbling râles over the lower third of the chest wall. In the early stages of primary pneumonia, high-pitched sibilant or fine crepitant râles are common; while in the advanced stages, with pleurisy, splashing, friction sounds, and a variety of musical râles are the rule. When consolidation is extensive but incomplete, the bronchi remaining open, one may hear bronchial breathing, also termed tubular or blowing breathing. This is comparatively common in calves, and is merely a continuation of the normal laryngeal or tracheal sound. It exceeds in intensity any other sound heard on auscultation of the chest. When consolidation is complete and the line of demarkation between the hepatized area and normal lung tissue is distinct, there may be complete absence of râles; this condition is common in calves. Infrequently consolidation is extensive and complete, massive pneumonia, when there may be complete absence of sound on auscultation. In acute types râles are most marked at the ventral border; in chronic types they may be heard anywhere; while in certain subacute to chronic sporadic forms in cows, distinct râles over the entire surface of both lungs may persist for weeks. On auscultation, the vesicular murmur, or respiratory sound, may show a distinct variation when the right and left lungs are compared; this indicates disease even though one may not interpret the sound.

Percussion induces pain in nearly all forms of pneumonia in cattle. Pain on percussion is less common in the horse, where it suggests pleurisy. Modified sounds in the form of dullness, or tympany, mark the location of an extensive consolidation; usually this is along the lower border of the chest. The interpretation of percussion sounds requires much practice. Cough on percussion often comes with the first blow, when it is positive evidence of pneumonia. An area of distinct dullness over the lower third to half of the chest wall, bounded above by a well-defined horizontal line, indicates an accumulation of fluid in the chest cavity. In this condition, dyspnea and a marked expiratory lift are usually present.

The *course* depends on the underlying disease, and it varies widely. The primary form may terminate in about a week, but it may continue indefinitely, and even become chronic. Deglutition pneumonia usually ends fatally in two to five days; rarely in cows it persists for months. In vagus pneumonia the course is chronic. In primary sporadic pneumonia the outlook is good, except in the young, even when extensive. Secondary pneumonia always presents a grave prognosis, and recovery is hoped for only when the invasion is slight and recovery from the primary disease is prompt.

Diagnosis.—From a diagnostic standpoint there are two chief problems in pneumonia: one is to recognize the disease, the other to determine whether it is primary, secondary, deglutition, verminous, etc. The disease is usually recognizable by one or more of the physical symptoms found on examination of the respiratory system; especially useful are increased rate of breathing, cough, râles, and cough on percussion over the chest. In conjunction with these, consider the severity of the general symptoms, the season, and the predisposing factors. In judging whether it is secondary in cattle, consider traumatic gastritis, mastitis, metritis, and any other pyemic condition that may exist. Pneumonia has often been mistaken for bronchitis. Cabot[11] states that "in the vast majority of cases of acute bronchitis, foci of bronchopneumonia are also present."

Chronic localized pneumonia in cows, resulting from incomplete recovery from calf pneumonia, or "hoof rot," may present a syndrome which leads to a diagnosis of traumatic gastritis. Early in the year a heifer that calved for the first time in the previous October, lost in condition, ate poorly and did not lie down when kept in a stanchion. There was a swelling of the left stifle and lameness. The respirations and temperature were normal, but the pulse was only 32, and remained there during the two months that the animal was under observation. The only respiratory symptoms were slight râles that could be heard only occasionally after repeated examinations. There was no cough and a cough could not be induced. Percussion revealed soreness over almost any part of the chest and abdomen, but it was especially apparent over the region of the liver. Pinching of the spine over the withers caused a marked and sharp reaction. Because of the symptoms and a statement by Wirth-Diernhofer[17] that a slow pulse may be caused by chronic peritonitis or chronic intestinal catarrh, a rumenotomy was performed but no abnormal condition was found. The diagnosis was then changed to multiple abscess formation, possibly of the liver, which might have resulted from chronic pododermatitis. A low grade chronic infection of the interdigital tissues was present and was suspected to be the cause of the swollen stifle. Blood examination gave neutrophiles

(segmentation) 87 per cent, normal 10-30, suggesting a chronic infection. The animal ate a little each day but finally was destroyed because of progressive and advanced emaciation. Autopsy May 7, 1952: "There were yellow fibrin deposits over the right parietal pleura. There was bronchiectasis in the right apical lobe of the lung, and the lobe contained light-green tinged pus. The right cardiac lobe was consolidated and the bronchioles contained pus. There were small amounts of pus in the posterior portions of the diaphragamatic lobe. Lung cultures: 1. *Actinomyces necrophorus*; 2. *Anaerobic streptococcus*. Local necrosis in the lungs of cattle, due to the necrosis bacillus, was described in 1890 by Bernhard Bang.[18]

Treatment.—General Care.—The first consideration is to provide a dry place, free from drafts. The value of open air has been highly rated, and justly so; but it is difficult to provide animals with open air in the cold seasons without exposing them to harmful cold drafts. The essential requirements are dryness, medium temperature (60°-65° F.), absolute rest, and nourishing food. Drinking of water should be encouraged by offering a fresh supply frequently. Cold damp weather is unfavorable to pneumonia, and removal of a patient from a cold damp stable to one that is dry and warm is almost invariably followed by improved symptoms. Open-air treatment is beneficial when the weather is suitable.

Since the introduction of sulfonamides, especially sulfamerazine, and sulfamethazine in the early Forties, there has been a sharp decrease in the severity, complications, and mortality in pneumonia. As reported by Roberts and Kiesel:[12]

"A review of the results of treatment of pneumonia in cattle for the past twenty years reported by the New York State Veterinary College Ambulatory Clinic, indicates a marked drop in mortality since the use of sulfonamide therapy. At the present time, the mortality in our cases of pneumonia has reached a low point of about 5 per cent.

"Treatment with sulfamerazine, sulfamethazine, and penicillin resulted in the recovery of 94.6 per cent of 129 cases of the pneumonic form of hemorrhagic septicemia in older cattle. This is a definite improvement over the results obtained by using the other sulfonamides.

"Treatment of 93 calves with the newer pyrimidines (sulfamerazine and sulfamethazine) and penicillin resulted in recovery of 81.7 per cent.

"Toxic reactions from sodium sulfamerazine and sodium sulfamethazine were not observed when used in 5 to 12 per cent solutions in the recommended dosage of ½ to ¾ grain per pound of body weight daily. In 150 cases of pneumonia and other conditions in which the sodium

salt of these drugs was used intravenously, there were only 2 instances where a toxic reaction was noted."

Using sulfamerazine, McAuliff[13] treated 27 cases of calf pneumonia and 122 cases of hemorrhagic septicemia pneumonia with a recovery rate in each of over 90 per cent. Christian[14] observed that in both cows and calves one grain of sulfamethazine per pound of body weight was effective when given orally once daily until temperature and respiration were normal. In severe acute conditions an initial dose of sodium sulfamethazine was administered intravenously. Of 43 calves treated, 42 recovered, and all of 8 cows treated recovered. No toxic reactions were noted, even when the dosage was double that recommended.

Because of the frequency of *Pasteurella* infection in pneumonia in cattle, antiserum has been widely used, but at present its chief use is to confer passive immunity to cattle about to be shipped.

Penicillin has recently proved to be the most effective remedy in the treatment of pneumonia. Because of the remarkable action of this drug and other antibiotics, their use in pneumonia has prevented many former complications and symptoms. Since *Pasteurella* is gram-negative and resistant to penicillin, and our knowledge of the infectious agent in the usual case of pneumonia in animals is unknown at the time of treatment, it is a common practice either to combine penicillin with other antibiotics, or to change from one to another when improvement is not apparent after 24 hours. Recommended dosage is from 1,000 to 2,000 units per pound daily, administered intramuscularly in one or two doses at twelve to twenty-four hour intervals either in oil and wax, or as procaine penicillin. In severe infections higher blood levels are attained from multiple injections of aqueous penicillin given every three to four hours. When there is no response to penicillin within twenty-four to forty-eight hours, the infection may be due to an agent that is resistant to penicillin (gram-negative), and success may be attained by a change to aureomycin or terramycin or streptomycin. In the treatment of stockyards pneumonia in cattle, Cromley et al.[15] have reported excellent results from terramycin given intravenously, 500 mg. per vein every twenty-four hours for a calf under 500 lb., and 1 gm. for animals weighing over 500 lb. For immediate action from aureomycin Burkhart[16] prescribes 5 mg. per lb. body weight daily per vein.

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ENZOOTIC PNEUMONIA OF CALVES

Etiology.—In large breeding herds calf pneumonia is often the most serious of the acute infectious diseases; it is less prevalent and less severe in small herds.

Calf pneumonia belongs to the group of diseases of animals that may be initiated by faulty hygiene, and that once established transmits infection by contact. The first to suffer are usually low in resistance and unable to overcome an attack from autogenous infections such as *Pasteurella* or *Corynebacterium pyogenes*, which may normally be present in the tissues. With the progress of the disease, however, such infections may achieve greater virulence, and may be readily transmissible from one individual to another. A similar condition is described in bronchopneumonia in man, where the chief offenders are reported to be pneumococci and *Streptococcus epidemicus*.

Housing.—*Poor ventilation* in calf barns is often an obvious cause in temperate climates. Where calves are stabled by themselves unprotected by the body heat of large animals a uniform temperature may not be maintained. Artificial heat may vary widely, unless controls are installed. This occurs in the night when there is a sudden fall in temperature accompanied by a high wind, or when the air is stagnant during a

warm humid period. There are few systems of ventilation that do not depend on human control. Especially undesirable are intakes without any deflector. When the air enters it may drop to the calves where a brief exposure to the draft may cause trouble. Air from intakes should be distributed by conduits in such a manner that it is diffused through the stable without creating obvious currents. This may be done by connecting the intake with a shallow trough 18 to 24 inches wide and with side walls from 3 to 4 inches high, suspended just beneath the ceiling. From this open conduit air pours over the sides and does not create a draft. The outtake should remove the air from near the floor; an outtake at the ceiling is not suitable for calf barns in cold winter climates. If the stable is heated the incoming air should pass through the heating unit before entering the stable. A desirable temperature is from 45° to 55° F. The chief atmospheric danger is from overcrowding, drafts, stagnant air, and too high a temperature when artificial heat is used. But regardless of sanitation, pneumonia may develop in an individual in excellent physical condition, and either gradually or rapidly spread to a large percentage of animals in the unit. Freedom from pneumonia in calves raised in sheds open on one side indicates that the bovine is an outdoor animal and that housing under accepted standards of sanitation predisposes to respiratory disease.

Chilling may result from open doors and windows, cold drafty stables, and walls of stone or concrete. While concrete walls and partitions are readily cleansed and disinfected, they radiate cold even when the room temperature is adequate, and calves will often lie directly against them. Regardless of care and housing, in herds where calf pneumonia is prevalent sudden changes in weather are accompanied by new attacks, and calves that are already sick become worse.

Close confinement in crowded pens with tight high walls is undesirable because a young animal takes its air from near the floor. If tight walls are considered desirable to prevent contact, they should be only high enough for this purpose, not much higher than the head of the calf. Such pens should be not less than 6 feet in width. The bringing together of a number of young animals, as practiced in breeding establishments, nearly always leads to some pneumonia, and when the sanitation is poor from atmospheric extremes, or overcrowding, the rate of the disease and the mortality are high. Once the disease is established it spreads through the group in the form of a contagion; it may be introduced with a sick calf. In general, however, the chief initial influence is poor atmospheric hygiene, just as in diarrhea it is improper feeding. Whenever a calf in a pen with others comes down with pneumonia, it may be taken for granted that the place is overcrowded. A floor space adequate for

healthy calves may be insufficient when respiratory disease appears. An allowance of 36 square feet for each individual is abundant.

Associated Diseases.—In calf pneumonia at the usual age of from 3 to 6 weeks, secondary diarrhea is not infrequent; it may immediately precede the respiratory symptoms, or appear somewhat later, and in some it is terminal. When diarrhea persists at this age one may suspect pneumonia. In diarrhea of the first few days of life, pneumonia is rare, and then only as a terminal or septicemic lesion. The term "pneumoenteritis" applies only to certain individuals in which pneumonia and diarrhea are both present. In most cases of calf pneumonia there is no diarrhea.

Age Susceptibility.—There is a definite age susceptibility to calf pneumonia which begins at about 3 weeks, increases up to 6 to 8 weeks, and then gradually declines to about 4 months. While calves of any age are susceptible, and an outbreak may occur in 6-month-old animals, one may usually regard a 4-month-old healthy calf as relatively resistant. As an outbreak progresses an individual may succumb to the disease at 10 days of age and present lung lesions that may have originated shortly after birth. In such infrequent cases the disease has the characteristics of a calf septicemia with localization in the lungs and perhaps in the digestive tract as well.

Season.—Usually it appears in November, continues intermittently through the winter, and is especially destructive in the spring if April and May are cold. From June to September it is infrequent. Yet outbreaks that are clearly contagious and apparently primary may occur in September under the best of housing and atmospheric conditions.

Infection.—Occasionally the only apparent cause is infection. The disease may appear in the summer under excellent hygienic conditions, and it may be introduced by the purchase of an infected calf. Many bacteria have been implicated, and the laboratory may report negative findings. In a given outbreak, lungs of calves that die after a few days may be negative on culture, while those from calves that have been sick three or four weeks may be heavily infected with streptococci. In these cases it is not assumed that the streptococcus has etiological significance. *Pasteurella* have been regarded as a frequent bacterial cause, and in our cases they have usually been found. Jones and Little[1] have described an outbreak in calves from which *Pasteurella boviseptica* "Type I" organisms were obtained from the lungs in pure culture. With these cultures it was possible to induce nasal catarrh in calves. The same organism was found in the nasal discharge of others affected with either a nasal catarrh or pneumonia. It is probable that certain calves are carriers. Smith[2] has described a fatal pneumonia affecting the living,

full-term or slightly premature calf caused by *Br. abortus* being brought into intimate contact with the alveolar walls through the amniotic fluid. He believed the slight lesions induced by it predisposed to various subsequent infections of the lungs. Bacteria to which pneumonia in the young has been attributed are: *Corynebacterium pyogenes* (Ward),[3] the colon bacillus, and others.[4]

A severe outbreak of calf pneumonia caused by *Corynebacterium pyogenes* has been described by Schmid.[5] In cultures from a number of calves the pyogenes was overgrown by streptococci and other bacteria. The infection was spread through the medium of dirty pails.

Mixed infections are frequent and in a given epidemic the lungs of one calf may yield pure cultures of *Corynebacterium pyogenes*, while those of another may yield pure cultures of *Pasteurella*. But since present knowledge of the bacteriology of the disease cannot be utilized in either diagnosis or control, these variations merely illustrate the nonspecific nature of the infection.

Experiments have been conducted to prove that calf pneumonia is a virus disease. Transmission experiments conducted by Baker[6] led him to the conclusion that infectious pneumonia and diarrhea in calves is caused by a filterable virus. From infected calves he obtained a bacteria-free agent, capable of passing a Berkefeld N filter, that produced pneumonia in white mice by intranasal inoculation. The agent obtained in mice produced disease when inoculated intranasally into calves; the disease was transmitted to calves by pen contact, and was similar in every respect to the natural infection. Since pneumonia and diarrhea in calves usually occur as pneumonia or as diarrhea and at different age periods, additional confirmation is needed to incriminate a virus in this important group of affections.

Morbid Anatomy.—In acute types the anterior lobes are consolidated and the entire lung may be diffusely congested. In less acute types various degrees of bronchopneumonia with lung abscess, necrotic foci, and purulent bronchitis are present. The extent of the hepatization in certain chronic cases is remarkable. The initial lesions are in the anterior ventral lobes. On postmortem examination of a calf that has died of pneumonia the ventral parts of one or both the anterior lobes are involved; usually the pneumonia is bilateral. Consolidation may extend to all of the ventral parts of both lungs. The surface of the consolidated part is uniformly dark red and the hepatization is usually complete; it is an extensive confluent pneumonia. As a rule there is no pleuritis and no thickening of the interlobular septa as in the pneumonia of hemorrhagic septicemia of cows. The cut surface is firm and moist. Small grayish foci may give to the cut surface a granular

appearance; these foci represent accumulations of leucocytes. The cut surface is moist and uniformly red or granular in appearance. In the bronchi are foam, mucopus, or mucus. The pneumonia is *exudative* in type, the exudate being composed of leucocytes, and red cells.

There may be some emphysema, and often there are various degrees of enteritis from catarrhal to hemorrhagic. Fibrinous pleuritis has been found on autopsy. Usually there is no fluid in the chest cavity. When there has been an abundant nasal discharge one may find a fibrinopurulent rhinitis and sinusitis.

Symptoms.—Attention is first drawn to the calf by dullness, cough, or fast breathing. On examination the conjunctival mucosae may be congested. The temperature is 103° to 106° F., and cough is easily induced. Temperature readings of apparently normal animals may reveal a fever one to two days before respiratory symptoms appear. Dullness, poor appetite, a rough hair coat, and rapid loss in condition are frequent. Yet the calf may continue to take milk until within a few hours of death. In the less acute forms the breathing and temperature may show considerable daily variation. A mucopurulent or purulent nasal discharge is occasional and when abundant it is a bad sign. On auscultation, high-pitched sibilant râles mark the less severe types, and coarse bubbling or bronchial sounds are of grave significance. Râles may be entirely absent in types that present on autopsy sharply defined areas of consolidation; in these cases the respiratory sounds may apparently be normal or greatly increased (bronchial breathing). Percussion may induce cough, cause pain, or infrequently reveal a dull area. As a rule the percussion findings are negative, though in fibrinous pleuritis there may be dullness over the entire chest wall. Diarrhea is occasional, but it has no relation to the diarrhea of calf septicemia occurring at an earlier age; when diarrhea persists at an age of from 3 weeks to 6 months one may suspect pneumonia. The most important diagnostic signs are fast breathing, induced cough, and high fever. When this combination exists, a diagnosis of pneumonia is always justified regardless of negative findings on examination of the chest. Rapidly fatal types are unusual. They are characterized by sudden prostration leading to death within twenty-four hours; the animal is recumbent and respiratory symptoms may be slight. Such cases are met with at the height of a severe epidemic and toxemia is the chief condition. A calf with extensive pulmonary lesions may be found dead from pneumonia without having shown symptoms that were recognized, even where daily temperatures were being taken in an effort to discover new cases. In an infected group, some remain well, some have only a bronchial catarrh, while others suffer from various degrees of pneumonia.

The *course* is indefinite. If improvement is not distinct at the end of two weeks, the condition is usually hopeless. Deaths are common within two to four weeks after the onset; they may occur earlier or the animal may finally die months later. Some appear to recover only to die later from a flareup of an old necrotic focus. A few recover completely. Usually, however, they either die early or after an intermittent or prolonged course, or they make an incomplete recovery and remain permanently stunted. It is not a disease of the newborn, though pneu-

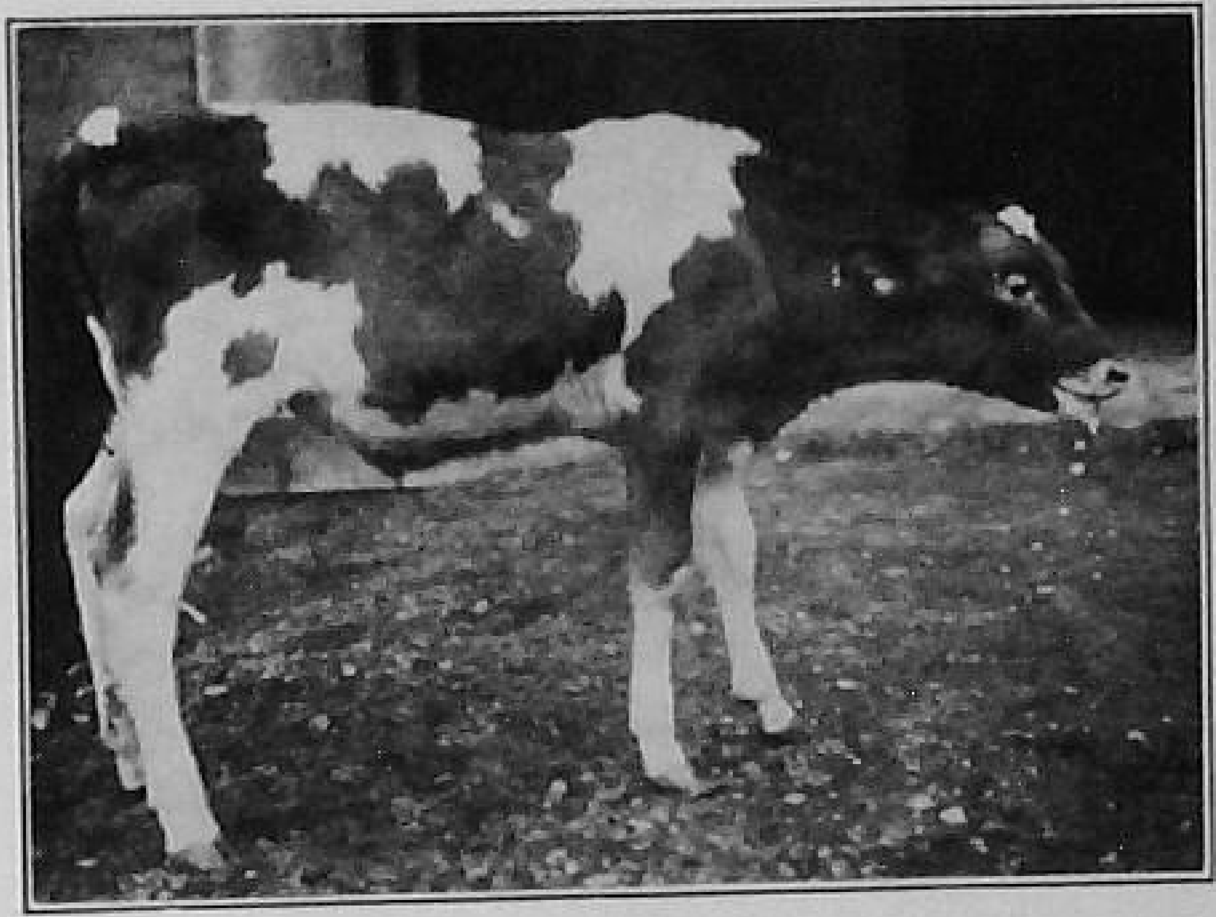

Fig. 2.—Calf pneumonia.

monic lesions in calf scours of the newborn are occasional. With few exceptions the mortality has been from 50 to 75 per cent, but this has been lowered since the introduction of antibiotics and sulfonamides. The prognosis is better in a small herd than where many calves are housed together in a single unit, and where there are frequent additions and removals. The mortality varies widely in different herds, as well as in the same herd in different years.

The *diagnosis* presents a few difficulties. Among calves and young stock, sudden rapidly spreading outbreaks of colds in the winter months are not infrequent. In a group of affected calves, there may be a few that breathe faster than normal and carry a temperature of 104° to 106° F. Some of these may be entirely normal in two or three days, while others have genuine calf pneumonia. In all such acute febrile respiratory attacks the diagnosis should be pneumonia. In calf septicemia or white scours, individual cases may present symptoms and lesions identical with those of acute calf pneumonia; and individual cases of calf septicemia may be left with a secondary chronic pneumonia, also identical in symptoms and lesions with those of chronic calf pneumonia. With few exceptions differentiation is not difficult because of the pre-

dominant early age—3 days, and diarrhea, in one group; and the predominant older age—3 to 6 weeks or more, and pneumonia in the older group. It is of course possible, that where hygiene is especially bad, both affections may prevail on the same farm. Pneumonia that is slowly chronic from the beginning is easily interpreted as a chronic cold or bronchitis; when in doubt, diagnose it as pneumonia. In all epidemics of colds, usually one or more animals are affected with pneumonia. There is always the possibility that in persistent diarrhea pneumonia may develop.

Treatment.—In severe types, when pneumonia is purulent, or extensive, or necrosis and abscess have developed, there is little hope for recovery. Early removal from damp or cold quarters to a dry warm place is often beneficial. Fresh air is desirable, but not at the cost of warmth, dryness, and protection against drafts. An excellent place for a calf affected with pneumonia is in an improvised small pen occupying the space of one or two stanchions in the cow stable. To protect against drafts, the sides of the pen may be covered with coarse sacking.

Since the introduction of sulfonamides and antibiotics, such as penicillin, the success which has followed their use in the treatment of pneumonia in general has been observed in the treatment of the young of all species, and especially in the treatment of calf pneumonia. In 1946 Schwarte and Biester[7] reported the value of penicillin in enzootic pneumonia in calves. A 6-weeks-old calf that had been severely sick for three days, was hardly able to stand and had a temperature of 106° F. It recovered completely after receiving a total of 780,000 units of sodium penicillin, given subcutaneously in divided doses every three hours over a period of ten days. As reported on page 46 recoveries from calf pneumonia have been over 80 and 90 per cent. At present, superiority in the antibiotics has been credited to penicillin, and in the sulfonamides to sulfamethazine and sulfamerazine. While penicillin has gained preference over all other remedies, and is described as a more potent antibacterial agent than the sulfonamides, it may encounter resistant infections (gram-negative) which succumb to other antibiotics, such as aureomycin, terramycin, streptomycin, chloramphenicol (Chloromycetin), or to the sulfonamides.

The accepted dosage for the sulfonamides (sulfamerazine, sulfamethazine) for the first one or two days is 1 grain per pound of body weight (6.5 grams per 1000 pounds) orally in two or three divided doses, followed by not more than one-half of this amount for not more than a total of five days. To avoid errors in dosage, administration should be in capsules or tablets which are conveniently given to calves with a small balling gun. To obtain prompt action in a severe acute

attack, administer sodium sulfamerazine or sodium sulfamethazine, ¾ to 1½ grain per lb. body weight in a 25% sterile solution, per vein, the first day, followed by ¾ grain per lb. body weight for the following three to four days. Or one may use equal parts of these two sulfonamides, solution of 5 per cent each (Merameth).

Prophylaxis.—Prevention is the most effective protection against calf pneumonia. Apply the general care outlined in the chapter on diseases of the newborn. Segregate the sick. This means that they should be permanently removed to a separate stable and cared for by attendants who do not enter the calf stables. Overcrowding should be relieved by separating the well from one another as widely as possible; this needs to be done with the appearance of the first case of pneumonia. Whenever a sick individual is removed from a group of calves, its empty place should not be used for additions. In time the remaining calves will outgrow the most susceptible age period—4 to 8 weeks, if no more young calves are added to acquire the infection and intensify its virulence. Whenever pneumonia appears it is imperative that stables not recently occupied by calves be found for the additions.

Various types of stables have been constructed to prevent pneumonia. The most effective consist of a number of small units, which provide conditions similar to those of small herds. Calf barns in use by the Animal Husbandry Department of the New York State College of Agriculture, for example, have dimensions of 18 by 24 feet. In each barn are eight stalls 6 by 6 feet square. Four small deflected air intakes open near the ceiling and a large outtake opens near the floor at one end. As a rule there is only one calf to a pen, though two newborn calves may be placed together. At the New Jersey Experiment Station[8] steel mesh floors have been installed on the principle that raising the calves above the concrete stable floor will prevent respiratory infection. The calves are tied in a single row far enough apart to prevent direct contact. The steel mesh or woven wire floors should be raised high enough above the floor to permit easy access for cleaning and drainage. With this system of housing, a minimum of labor is required for feeding and care. Under both of these systems individuals are separated from direct contact with each other, but on the wire mesh floors there is a greater concentration of individuals in the stable. The open-pen method of raising calves, as described on page 169, Fig. 19, has completely prevented calf pneumonia in certain large herds where it had caused heavy loss for years.

In the use of separate stables, it is desirable to fill a unit with a group of newborn calves, and make no more additions until after the group has been removed to quarters occupied by older stock. By prac-

ticing this rotation of groups, calves of the age most susceptible to pneumonia will not be distributed through all the different units at one time.

Prophylatic vaccination with freshly prepared *Pasteurella* bacterin has apparently been effective in groups where this type of infection was active; in some large herds it is used as a routine at birth. Give 1 cc. the first day, 2 cc. the second, 3 cc. the third, and 4 cc. the fourth. It has been given in this manner up to 10 cc., but the higher dosage sometimes causes sudden deaths. Jones and Little[1] gave two injections of killed cultures of "Group I" hemolytic *Pasteurella* to calves that subsequently developed rhinitis, and yielded "Group II" *Pasteurella* on culture. Hemorrhagic septicemia aggressin is also used, but it seems to be less satisfactory than a freshly prepared bacterin. While prophylactic vaccination usually has no apparent effect, it is generally regarded as useful, especially when autogenous killed cultures are used. The most effective bacterins are those prepared from cultures obtained from the infected herd, and these will prove worthless against kinds of bacteria whose activity does not establish immunity. At best, it is a form of insurance of uncertain value. As in all other diseases of the young, strict observance of the principles of good hygiene is the most effective protection against loss.

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PNEUMONIA IN PIGS

Etiology.—Exposure to cold, moisture, and filth is a cause of highly prevalent pneumonia in pigs and young swine, which occurs in the absence of such specific infections as hog cholera, swine influenza, and lungworms. In large herds it is commonly enzootic, occurring in

twos and threes, while in small groups it may affect only single animals. It is especially prevalent in piggeries where the housing is insufficient, the ventilation poor, the construction of concrete, or the bedding scanty. As in pneumonia of calves, it may be associated with enteritis. Exposure is intensified in swine by the habit of pigs to gather in heaps; this causes overheating in the center, while those on the surface may be shivering with cold. On scattering, the overheated pigs are soon chilled. Exposure during transportation is also a frequent cause of pneumonia in swine.

Bacteriology.—In pneumonia caused by exposure a variety of organisms have been found, either in pure or mixed cultures. In pneumonia associated with enteritis, for example, McBride[1] examined lungs from 13 cases in a large garbage-feeding plant where it was thought the loss might be due to hog cholera resulting from so-called "breaks" in immunity. Pure cultures of *Salmonella suipestifer* were recovered from 7, and pure cultures of *Pasteurella multocida* from 3. He concluded that "the condition was primarily an enteritis incited by *S. suipestifer*, with later involvement of the lungs, and in the majority of cases the condition might be termed pneumoenteritis." Streptococci and *Corynebacterium pyogenes* are found in subacute and chronic forms; they colonize in the lungs during the later stages of the disease. Mixed infections are the rule in enzootic pneumonia of pigs. Jones[2] has reported that "swine plague or hemorrhagic septicemia" is a serious disease of swine in the Province of Ontario where it occurs most commonly in fall and winter litters and chiefly affects young pigs shortly after they are weaned. He emphasizes the influence of anemia while nursing the sow, shipping, exposure to cold and rain, damp pens, removal from the warmth of the sow, and a change of diet; influences which lower the resistance are necessary to initiate the pneumonia. Schofield[3] writes that in Canada hog cholera is rarely seen, while virulent outbreaks of septicemia due to *Past. multocida* are of common occurrence. Hopkirk[4] states that in New Zealand *Pasteurella* and *Salmonella* infections are responsible for a large proportion of cases of pleurisy and pneumonia in pigs, and that field experience has shown that these conditions are associated with bad feeding and housing, and will disappear largely when these factors are attended to. Birch and Benner[5] have reported that *Pseudomonas pyocyaneus* infection may spread slowly through a herd of young swine, causing heavy losses from pneumonia, but apparently this is infrequent.

Morbid Anatomy.—In the lungs there are various degrees of congestion, consolidation, edema, and pus formation. Pleuritic adhesions are not infrequent. The trachea and bronchi may contain fibrinous

exudate. The submaxillary lymph glands are swollen and congested. Enteritis is not infrequent.

Symptoms.—The attack may be acute, but usually it is either subacute or chronic. At first the pig is listless and does not eat. Efforts to avoid examination lead to dyspnea, thumps, and the pig soon gives up from fatigue. The temperature is variable, but usually there is a medium to a high fever. Cough is easily induced, and there may be a mucous discharge from the eyes and nose. Breathing is fast and râles may be heard on auscultation over the lungs. The mortality is high. Since the exact nature of the infection cannot be learned promptly by the clinician, it is good judgment to consider all cases of pneumonia as contagious.

Diagnosis.—Swine influenza may be suspected when a number of pigs are attacked shortly after possible exposure. This occurs on return from a fair and other outside contacts and in areas where swine influenza is regularly present. Lungworm disease is readily detected by means of a complete postmortem examination of the lungs. Finally, pneumonia may be a lesion of hog cholera, or of any other general infection, or it may be secondary to enteritis.

One needs to determine whether the pulmonary lesion is secondary, possibly to infectious enteritis, or has resulted from the direct effect of exposure to cold and moisture and poor housing. The present tendency is to regard all contagious pneumonia in swine as hog cholera or swine influenza or lungworm disease, and all other pneumonia in swine as secondary.

Treatment.—In the treatment of 380 animals aged 8-10 months for enteritis-pneumonia with sulfamerazine and sulfathalidine, Edmonds[6] has reported a recovery of 88 per cent. In 26 untreated controls the mortality was 100 per cent. In the early stages of the disease, sodium sulfamerazine 3 Gm. daily per 85-100 lb. body weight in 6 per cent solution intraperitoneally, in a few cases subcutaneously, gave prompt and beneficial effect. Maintenance dose of sulfamerazine was 1 Gm. per 10 pounds body weight daily for one or two days and then reduced by 50 per cent. Sulfathalidine, 1 Gm. per 10 lb. body weight daily, continued for several days and reduced with improvement was found to be adequate. For administration of the 4-gram bolets of sulfathalidine orally, preference was given by Edmunds to British capsule forceps and a swine mouth speculum. With the use of a balling gun the pharynx may be injured, or the tablet may be deposited in the pharyngeal recess.

Fox and Burkhart[7] have reported successful treatment with sodium sulfamethazine in a herd of 400 garbage fed swine where 80 pigs had died. In 32 affected with pneumonia a diagnosis of pasteurellosis was

made on typical symptoms, postmortem findings, and the isolation of *Pasteurella multocida*. The treatment consisted of subcutaneous injection of 25 per cent sterile solution sulfamethazine at the rate of 1½ grains per pound body weight the first day and 1 grain per lb. body weight on the second and third days; this treatment provided adequate therapy when administered early in the attack.

Harms and Langer[8] obtained similar results from sulfamethazine in a mash, 1½ grains per lb. body weight the first day, 1 grain the second and third. More prompt improvement followed the use of sodium sulfamethazine, 25 per cent solution, 1 grain per lb. body weight intraperitoneally for three days.

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SHEEP PNEUMONIA

While pneumonia in sheep is similar in pathology and etiology to that in swine and calves, apparently it is less frequent. In Colorado Newsom[1] has written on that form of pneumonia resulting from exposure, which "affects sheep of all ages in all climates, beginning with lambs just after they are dropped and continuing through to the feedlot, and to the farm or range." Among the chief causes are exposure after shearing and dipping, transportation (chilling, fatigue, fasting), and storms even in the summer where sheep are pastured at high altitudes. Newsom and Cross[2,3] concluded that ovine pneumonia may be caused either by *Past. multocida* or *Past. hemolytica*, and wrote in 1932 that "our unpublished observations do not support the view that *Past. oviseptica* can be regularly isolated from a great variety of diseases." Montgomerie, et al.,[4] encountered an outbreak of acute pneumonia in sheep, mostly in mature animals in North Wales in May when an ex-

tensive heat wave was followed by cold east winds; the mortality was rarely over 15 per cent of the flock; they also found *Pasteurella* similar to that described by Dungal[5] in Iceland.

The *postmortem* changes are similar to those of acute pneumonia in other species: consolidation of one or both lungs, pleural effusion, and pleural hemorrhage; usually it is a diffuse lobar pneumonia.

Symptoms.—It begins with depression, anorexia, drooping ears, often a discharge from the eyes and nose, cough, respirations 50-60, and a temperature of 106°-107° F. The usual course is from 5-7 days, but death may occur as early as 12 hours after the onset. "Most animals with well-developed pneumonia will die, although some recoveries do take place."[1] In a car-load of "feeder" sheep that were sheared and shipped from Utah to New York, the mortality from "shipping disease" in transit was nearly 100 per cent, regardless of intervals of unloading for rest and feeding and removal of the sick.

Treatment.—In the treatment of feed-lot lambs affected with pneumonia, Forsyth[6] has reported success with the following method. Segregate those that are eating and give sulfanilamide in the grain at the rate of 1 grain per pound of sheep per day. The sheep that are off feed are treated with sodium sulfathiazole at the rate of 50 grams per 5 gallons of water. At the end of three days most of the sheep will recover. Sulfamerazine, as well as penicillin, are also indicated.

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CHRONIC PROGRESSIVE PNEUMONIA IN SHEEP

(*"Lungers"*; *Jaagsiekte*, S. Africa)

Since 1915 a fatal chronic pneumonia in sheep has been recognized in Montana, Oregon and other Northwestern States, and an apparently identical form also occurs in South Africa. It is characterized by an insidious onset and a gradually progressive dyspnea leading to emaciation and death. Losses in Montana have been estimated at from 2 to 10 per cent of affected herds. Older sheep suffer chiefly, but younger

animals are susceptible. In 1923 Marsh[1] reported that the existence of the disease was first brought to the attention of the Montana Live Stock Sanitary Board in 1915, and that he believed it to be an independent affection. The disease in England has been described by Blakemore and Bosworth,[2] and in Iceland, where it has caused heavy loss, by Dungal, et al.[3,4]

Etiology.—The cause is unknown. Transmission experiments have been conducted by Creech and Gochenour.[5] Working with 23 diseased and 96 normal sheep they were able possibly to transmit the disease to 4 animals by intrapulmonary inoculation of emulsified lung tissue. On bacteriological examination they found pure cultures of *Pasteurella* and of *Corynebacterium ovis* in some cases. But since agglutination and complement-fixation tests were negative, these microorganisms were regarded as of no relation to the cause. There is no evidence of direct transmission.

Morbid Anatomy.—The postmortem changes are limited to the thoracic cavity. When the chest is opened the lungs fail to collapse and completely fill the thoracic cavity. Pleuritic adhesions are frequently present. Consolidation is usually extensive, though it may be incomplete. The cut surface is gray and coarsely granular. The bronchi contain a small quantity of mucopurulent material. The bronchial and mediastinal lymph glands are swollen and edematous. In the descriptions of the histopathology by Marsh,[1] and Creech and Gochenour,[5] emphasis is placed upon the presence of epithelial proliferations of the alveoli and bronchioles, and upon the extensive fibrosis, changes which are regarded as characteristic of progressive pneumonia.

Symptoms.—The onset is so gradual that it is not known how long the animal has been affected when the symptoms are recognized. When driven, affected sheep lag behind the herd and breathe heavily. Somewhat later expiratory dyspnea is constantly present. Finally, breathing becomes rapid and labored at all times and mouth breathing appears. The nostrils are dilated, but cough and nasal discharge are not usually present. The temperature is normal. The course is over a period of weeks. The mortality is 100 per cent.

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INHALATION PNEUMONIA

(Aspiration Pneumonia; Deglutition Pneumonia; Mechanical Pneumonia; Pulmonary Gangrene)

This is a highly fatal type caused by inhalation of gases, food, or drugs. It may often result from faulty administration of medicine in a drench, usually given by the owner or attendant. The head is fixed and the drench is literally poured into the lungs. Especially dangerous are drenching through the nose, and drenching a recumbent animal. Swine are subject to inhalation pneumonia through their violent opposition to the administration of medicine in the form of a drench. Since the introduction of the stomach tube for giving bulky liquids, this form of pneumonia has diminished. Inhalation pneumonia in calves and swine has resulted from aspiration of food when eating greedily. In the cow inhalation pneumonia has been caused in choked animals by administration of a drench to relieve bloat, and this accident is nearly always fatal. Foreign body pneumonia in sheep from inhalation of timothy hay and pieces of hay and straw is not infrequent and readily occurs in sheep fed on cut hay. Horses suffering from pharyngitis, or catarrh of the upper air passages, easily contract pneumonia when irritant drugs are given forcibly with a dose syringe. It sometimes follows chloroform anesthesia; this may come from prolonged irritation of the mucosa, or from eating while the throat is still paralyzed. In the horse it may follow a throat operation, or inhalation of pus from a ruptured retropharyngeal abscess. In cows, it has resulted from injury to the throat from whipstocks and other probang substitutes used by the owner. It may be secondary to inflammatory or paralytic conditions of the throat, as seen in encephalitis, milk fever, pharyngitis, choke and esophagitis. Occasionally a choked animal is drenched with oil for the purpose of lubricating the obstruction; this practice nearly always results in fatal pneumonia. Inhalation of smoke, fire, and fumes is an infrequent cause.

In chronic lead poisoning in equines there is a paralysis of the throat which results in deglutition pneumonia and pulmonary gangrene; this is described under lead poisoning.

Morbid Anatomy.—When death occurs in a cow after a course of not more than forty-eight hours, one may find diffuse serofibrinous pleuritis with an abundance of fluid exudate in the pleural cavity. The anterior ventral parts of the lungs are solidified and on section there

is found an acute hemorrhagic pneumonia with some interlobular edema, and a slightly fetid odor. When death occurs a few days later one finds abscess formation, necrosis, gangrene, and extensive emphysema.

When death occurs in a horse after a course of about three days one may find the ventral lobes of both lungs dark red, firm, and consolidated. On section, the cut surface presents dark and gray areas surrounded by deeply congested tissue. In the apical lobes there may be foci of necrosis. After a course of not more than four days there may be complete consolidation and gangrene of as much as the ventral third of one or both lungs. If the course has been somewhat longer the pleural cavity may contain an abundance of brownish yellow fetid fluid. Masses of fibrinous exudate may cover the ventral surfaces of the lungs and the adjacent parietal pleura. On section, the lung tissue is dirty reddish brown and gangrenous, and the bronchi contain reddish brown fetid material of the consistency of mucus. The pericardial sac contains more or less serous exudate. The extent of the gangrene and necrosis is in direct proportion to the length of the course.

Symptoms.—There is a history of a condition for which a drench may have been given, such as indigestion, colic, or acetonemia. Usually this is recent, not more than one to three days previous to the appearance of the symptoms of inhalation pneumonia. In the case of a cow, however, the veterinarian may not be called until after cough, fast breathing and unthriftiness have existed for weeks following the administration of a drench by the owner. Knowledge of previous drenching is sometimes withheld by the owner or caretaker to avoid blame for the death of the animal. The following illustrations show that the disease varies according to the species affected, the manner of drenching, and the material given. There are also differences in the symptoms for which the reason is not apparent. In horses and swine the mortality is high; in cattle, recoveries are much more frequent.

Twenty-four hours after a drench was given to a choked cow the following symptoms were observed: marked dullness, respirations 49 with open-mouth breathing, pulse 85, temperature 101.2° F., and a slight nasal discharge. Other symptoms were induced cough, coarse bubbling râles over the lower third of the chest wall, marked increased vesicular murmur over the upper two thirds, and suppressed heart sounds thought to be due to collection of serum in the pleural cavity. Death occurred on the second day.

Two days after a cow was drenched with soda because of "indigestion," she showed depression, pulse 78, respirations 50, and a temperature of 103.5; râles were present over the ventral borders of both lungs.

On the third day, breathing was distressed, the pulse 100, the respirations 90, and the temperature 105.4. Distinct bronchial or tubular breathing could be heard over the ventral parts of both lungs. On the eighth day the breath was fetid, the temperature normal, the pneumonic symptoms distinct, and the prognosis apparently hopeless. This animal finally recovered.

Twenty-four hours after a cow was drenched with molasses which caused violent coughing, she was found with a pulse of 104, respirations 46, and a temperature of 106.8. Percussion over the lungs induced cough and pain. On the third day splashing sounds in the pleural cavity were heard. On the fifth day the cow was emaciated and pleural friction sounds were recognized. Three weeks later the respiratory sounds were normal and there were no further symptoms of pneumonia.

Within twenty-four hours after a drench reaches the lungs of a horse râles may be heard, and after two to three days symptoms of pneumonia are distinct. On examination one finds a fast pulse, a normal or slightly elevated temperature, congested mucous membranes, increased frequency of breathing, sweetish breath, and a slight nasal discharge that may be tinged with blood. Râles are present, usually over both lungs. Usually death occurs within four to seven days after the administration of the drench.

Ten days after a drench was given by the owner to a 9-year-old mare there was a pulse of 80, respirations 34, temperature of 103, and congestion of the mucous membranes. Edema was present along the abdomen and on the hind limbs, and the animal moved only after persistent urging. The breathing was abdominal. Loud bubbling râles were heard over the lower half of the right lung. Death occurred two weeks after drenching.

In the diagnosis one is aided by information concerning a previous disease. Often the brief course, ending in death, helps to distinguish this type of pneumonia from that following exposure or infection; this is especially true of equines. More difficulty is associated with the diagnosis in bovines, where sporadic pneumonias are far more frequent than in the horse and where the course is less definite. Regard for the fact that sporadic pneumonia is infrequent in the warm months of summer may be useful. The sweetish or fetid breath, characteristic of gangrene, may be pathognomonic when it appears early. Another form of pulmonary gangrene occurs as a terminal condition or sequella in contagious equine pneumonia, and does not appear until the seventh to the tenth day. Pulmonary gangrene also occurs in chronic lead poisoning in the horse. The presence of coarse bubbling râles over both sides of the chest, along the ventral border, is highly suggestive, and

these râles are usually present immediately after the fluid reaches the lungs. In the horse the disease is highly fatal. In the cow, recovery may occur even after the condition is apparently hopeless. Similar to pneumonia in general in bovines, the course is indefinite.

The *treatment* of inhalation pneumonia is entirely symptomatic, and is described under the treatment of bronchopneumonia (page 46).

PULMONARY ABSCESS

(Suppurative Pneumonia)

Etiology.—Abscess of the lungs is a common fatal complication in pneumonia. Sometimes it is found in cattle and sheep as an apparently independent disease of the lungs; yet in these cases it probably originated either as a metastatic or a primary pneumonia, a parasitic invasion, or a perforation—traumatic gastritis. As a metastatic infection it may result from mastitis, metritis, navel-ill, or scours. This view is supported by the presence of abscesses in the liver, peritoneum, and pleura, as well as in the lungs; by the history in the case of metritis a year previous; and by observation of acute metritis that terminates in fatal pulmonary abscess. A strangles abscess may remain quiescent in the lungs for months and then extend rapidly. Pulmonary abscess has been observed in association with a thrombus in the right heart, and it occurs often in pulmonary tuberculosis and in traumatic pleuritis. Bacteriological examination of the abscesses may be negative.

Morbid Anatomy.—In the pyemic type the lungs may be so filled with abscesses that little normal tissue is found. Often the trachea and bronchi contain blood clots, and the lung tissue may be infiltrated with blood. Hepatic abscesses are not infrequent. In traumatic pleuritis the involvement is somewhat circumscribed.

Symptoms.—In cows the initial symptom may be dullness, or bleeding from the nose. The hemorrhage is caused by erosion of a pulmonary vessel, is either slight or abundant, and often it is foamy. Usually the condition of the animal is poor. The temperature is high, 104 to 106° F., the pulse 60 to 80, and the breathing fast. Food and water are refused for a time. Cough is present and may be induced. Over the lungs one hears a variety of râles. Percussion over the chest induces pain, and it may reveal areas of dullness. After the distinctive symptoms appear, a progressive loss in strength and condition leads to death in from two days to about three weeks. When the first symptoms are a loss in condition, appetite, and milk flow, one suspects either chronic traumatic gastritis, or tuberculosis. The emaciation, arched back, stiffness, and soreness on percussion over the chest also suggest traumatic gastritis.

But when blood appears at the nostrils the nature of the disease is revealed, though hemoptysis may not occur. When lung abscess becomes prominent during the course of another disease, there are distinct general symptoms of septicopyemia. Pulmonary abscess may develop in cows without causing physical symptoms.

Lung abscess in the horse may be a terminal condition in pneumonia. The pneumonia symptoms are prolonged, a chill develops, the condition becomes worse, and pus may appear at the nostrils. In diseases of the newborn, pulmonary abscess may be embolic in all species. The disease is incurable.

In a 2-year-old heifer that had been losing condition for three months, but had neither coughed nor breathed abnormally, there were found a large abscess in one lung and thrombosis of the hepatic artery.

An 8-year-old cow suddenly discharged a large amount of fine foam from the nose and open mouth. Breathing was fast and distressed and accompanied by an occasional grunt. Cough could not be induced. Many coarse bubbling râles were heard over the dorsal surface of both lungs, and there was a sound of fluid moving back and forth in the bronchi and trachea. Prompt relief followed the administration of adrenalin. But two days later the breathing was slow and labored, crepitating and moist bubbling râles were abundant, and there was no general improvement. Six days later subcutaneous emphysema had become general and the animal was destroyed as worthless. Autopsy revealed multiple abscesses in the ventral part of the anterior lobes, extensive pleuritic adhesions, and pulmonary emphysema. In this case the pleuritis and abscess formation may have been metastatic from metritis, or a result of either infectious or inhalation pneumonia.

LUNGWORM DISEASE

(Verminous Bronchitis; Verminous Pneumonia; "Hoose")

Definition.—Lungworm disease is an enzootic bronchopneumonia due to the presence of round worms in the bronchi. It is fairly common in sheep, calves, and swine. Goats often harbor the parasites, but usually they are not affected by their presence.

Etiology.—Lungworm disease is worldwide, but chief losses from it have been reported from the tropics, and from countries where the winters are mild. The incidence is reported as high in the lowlands of West Virginia and in California. It is common in Great Britain and Norway. And a letter from Quito, Ecuador, reports verminous bronchitis or hoose as one of the most dreadful diseases in that part of the country; attacking calves up to one year of age it has caused a mortality of 60 to 90 per cent; treatment has been useless and the disease

has been reduced only by indoor feeding. Exact knowledge is lacking on the prevalence of this disease in different parts of the world, but wherever it is common as a clinical affection it is serious. Weather often determines its prevalence in any locality, and where it is sporadic a wet season may precipitate a general attack. Westerheim[1] has reported from Norway that "in its humid atmosphere lungworm disease occurs frequently in goats and almost every old animal either harbors parasites or shows injuries caused by them. . . . Kids that were kept indoors all of the time with their heavily infected dams were not affected. . . . In the acute outbreaks, which are of infrequent occurrence, only a few of the flock become suddenly attacked, but many of the affected animals die."

According to Schwartz,[2] permanent pastures commonly cause a heavy infection in pigs. The humus and manure on such pastures favor the presence of earthworms and provide favorable conditions for the survival of eggs and larvae of other parasites that infect swine.

While it is chiefly a disease of growing animals, young adults are sometimes affected, and a few attacks in 2-year-old heifers have been observed. Serious outbreaks in adult dairy cattle in Great Britain have been reported by Smythe.[3] In temperate climates it appears as an enzootic shortly after the young stock are turned to pasture, reaching its height in July and August. Where there are no destructive frosts the disease may prevail the year round, as in calves in southern England.

An attack in calves does not confer immunity against subsequent exposure, according to Schmidt[4] of Giessen, who observed recurrence of the disease in cattle that suffered from an attack in a previous year and in another pasture. Kauzal,[5] on the other hand, has reported increased resistance in young lambs under 2 months old exposed to repeated daily doses of from 50 to 100 larvae of *D. filaria;* young lambs exposed for the first time exhibited remarkable variation in individual susceptibility, while lambs from 5½ to 7 months old had a much higher degree of resistance to first infection than younger animals. Resistance was thought to be due partly to age, and partly to previous infection; it was not obviously affected by deficient diet nor by concurrent infection with *Haemonchus contortus*. There is a definite age resistance which may be overcome in some individuals by massive infection.

Unthriftiness has been emphasized by most writers as an influence in the cause of lungworm disease. Thus Freeborn and Stewart[6] have written that in sheep it depends on a debilitating condition and does not occur in well-nourished animals. While the same principle may apply to some extent to cattle, there are many exceptions. On the few

farms where I have observed it in calves they were well nourished; in countries where the incidence in cattle is high it prevails widely in calves and yearlings in excellent physical condition, and it has been reported by Eveleth[7] in Arkansas in well fed sheep, goats, cattle, and swine. It is probable that the degree of infection has an influence. The disease may occur on farms not previously known to have been in-

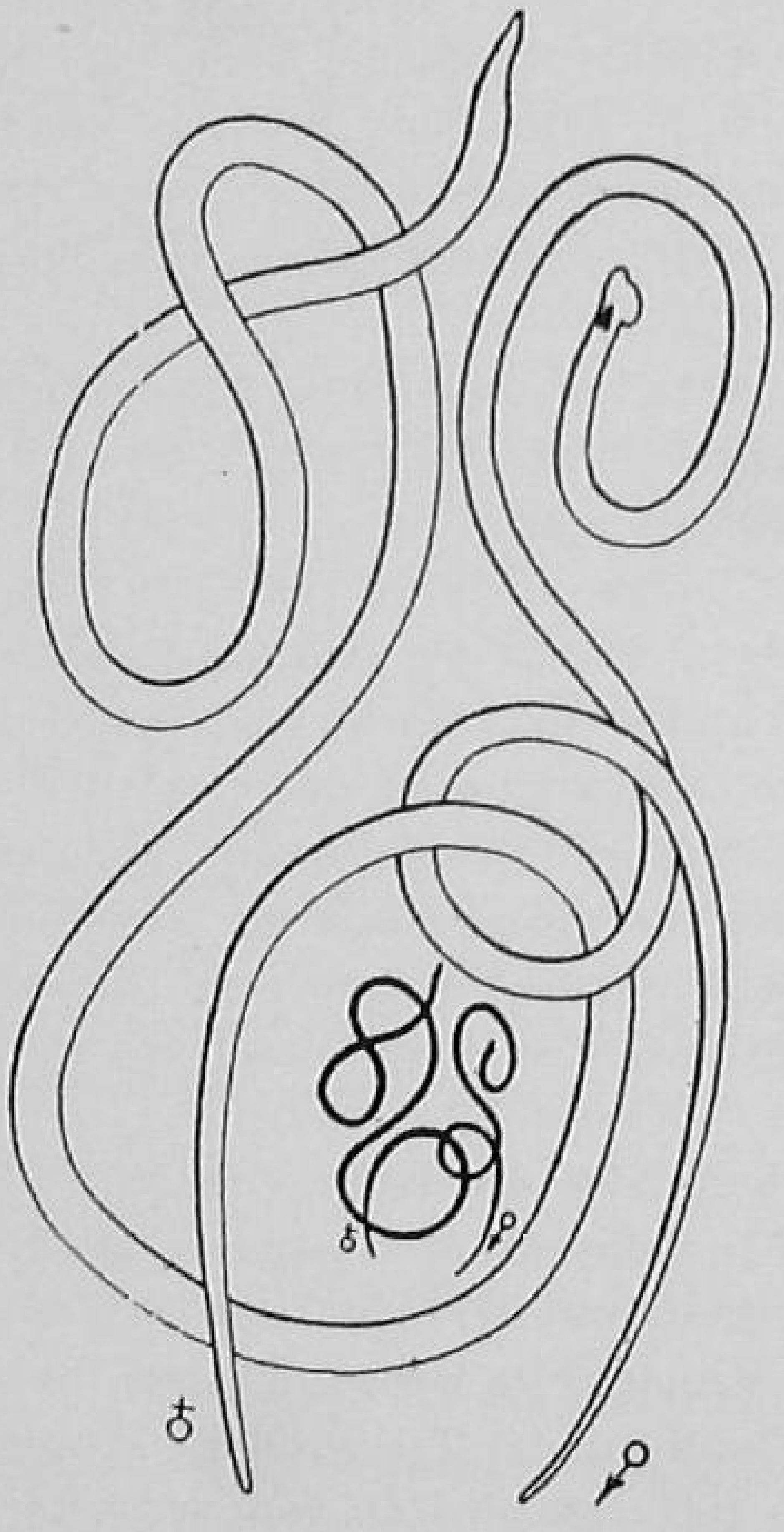

Fig. 3.—*Dictyocaulus viviparus*. Male and female. Small figures, natural size, large figures, x 5. From Neveu-Lemaire, 1918.

fected; it may disappear after taking several young animals in a single season; or it may recur year after year.

Parasitology.—In cattle the common lungworm is *Dictyocaulus viviparus* (male 3-8 cm., female 5-10 cm. long). The habitat is in the bronchi and trachea. Cattle may also become infected with *Dictyocaulus filaria* from sheep, goats or deer.

In sheep and goats the chief lungworm is *Dictyocaulus filaria* (thread worm). Male 3-8 cm., female 5-10 cm. long; color yellowish white with a brownish stripe. The habitat of the mature worm is in the bronchi

and trachea where they are grouped in coils. The eggs are deposited in the bronchi and they may hatch there in a few hours, but usually they are brought to the throat in mucus and swallowed. Unhatched eggs are rarely found posterior to the rumen. Guberlet[8] has reported finding the eggs in the small intestine, but not in the large. Larvae which pass out in the feces are about 0.5 mm. long; they may be identified by a small knob at the anterior extremity (Fig. 4). On leaving the host they become ensheathed, infective, and motile in from eight to ten days. They are now resistant to dryness, but are destroyed by disinfectants or freezing. The longevity of infective larvae is believed to be less than six months. They thrive in moisture and may develop

Fig. 4.—First stage larva of the large lungworm of ruminants, *Dictyocaulus* spp. (Courtesy of D. W. Baker).

in pools. They climb up on moist grass or roughage and as it dries they return to the soil. Thus the most favorable time for infection is when the grass is moist with rain or dew.

There is no intermediate host. Within three days after ingestion the infective larvae penetrate the wall of the intestine and enter the mesenteric lymph glands through the lymph vessels. Here they develop further and in about ten days after ingestion arrive in the lungs through the circulation. Intrauterine infection may also occur. The life history has been described by the Hobmaiers.[9] Guberlet[8] has described a lamb that received orally a capsule of embryos on January 25, it sneezed and coughed on February 18, and larval lungworms were found in the feces on March 4. Autopsy on March 21 revealed congestion of the extremities of the lobes of both lungs, and 175 worms, many of which were mature, were taken from the air spaces. In calves larvae appear in the feces as early as 20 days after experimental infection, but they disappear after about a month; the life of the adult in the bronchi is short. Under field conditions, lambs may remain a carrier of lungworms

and a source of contamination to a pasture for at least four months—Eveleth.[7] These parasites suck blood, irritate the mucous membranes, and cause bronchopneumonia. Larvae in the intestine may irritate the mucosa and cause diarrhea, but the chief damage is in the lungs.

Immunity.—In sheep the flock becomes immune within six weeks after the onset of an attack.

Mode of Infection.—Susceptible animals are infected by ingestion of contaminated wet grass, recently cut wet roughage, or water from contaminated pools or troughs. Fields and water are infected by feces from the sick, as well as from normal adult carriers, such as cattle, sheep, goats, or deer. It is probable that damp bedding in barns or sheds may harbor infective larvae and contaminate the food or water. In climates where the larvae are not destroyed each winter by frosts, chief importance is attached to grazing on wet grass as the manner of infection. I have found no report of infection from grazing on fields that are habitually dry, or from eating dry roughage, or that larvae will survive freezing.

Freeborn and Stewart[6] have reported that "in California infections occur most often when sheep share the range with deer," and that "where sheep and cattle shared the same pasture we have never taken the cattle lungworm, *D. viviparus* from sheep, but we have repeatedly taken *D. filaria*, the lungworm of sheep and deer, from calves." In one herd seen by the author, where several calves had died, goats were apparently the source of the infection. They occupied a small pasture in common with the calves, and no further trouble developed after their removal.

In sheep and goats the *hair lungworms* are represented by *Muellerius capillaris* (male 12-14 mm., female 33 mm. long) and *Protostrongylus (Synthetocaulus) rufuscens* (male 16-28, female 25-33 mm. long). The hair lungworms are reddish in color and are found in the smaller bronchioles and lung tissue; usually both species are present. The Hobmaiers[9] have demonstrated that the snail is used by these parasites as an intermediate host. Freeborn and Stewart[6] report that *P. rufuscens* produces extensive lesions and is one of the most important lungworms. While doubt has been expressed, by both Mönnig[10] and Cameron,[11] that the hair lungworms cause clinical symptoms, there is no doubt that they may occasionally cause clinical disease. Whether the parasite attacks the lung tissue directly, or causes a secondary bacterial infection is of minor importance.

Swine are usually infected with two species of lungworms: *Metastrongylus elongatus* (male 25 mm., female 58 mm. long) and *Metastrongylus brevivaginatus (Choerostrongylus pudendodectus)*, male

16-18 mm., female 19-37 mm. long. Both are common in the United States. The Hobmaiers[9] have reported that the unhatched eggs emerge from the hosts by way of the alimentary tract and are taken into the bodies of earthworms where they develop into larvae; in the earthworm they may survive the winter. Kates[12] has reported that in Beltsville, Md., some eggs in feces survived 381 days when buried 6, 8 and 12 inches, but that most of these eggs succumbed in about 290 days. They are ingested in earthworms by swine, enter the circulation from the intestine, and are deposited in the mesenteric lymph glands, where they undergo further development. From here they again enter the circulation and are carried to the lungs, where they reach maturity in about 4 weeks. These observations have also been reported by Schwartz and Alicata.[13] Swine-lungworm larvae are carriers of the virus of swine influenza. Elimination of eggs is especially abundant from young pigs. If young pigs are lightly infected they develop a resistance against subsequent exposure. There is an age resistance, but only because of a previous infestation from which they have recovered, according to Schwartz.

Morbid Anatomy.—Often the cadaver is emaciated and the perineal region may be covered with feces from diarrhea. The external surface of the lung may be normal, but as a rule one notes patches of pneumonia or atelectasis. On opening the trachea and bronchi the mucosa is found to be red and inflamed. In the bronchial tubes are many or few lungworms. These are often present in enormous numbers, nearly filling certain bronchi, or thickly clumped together in a few of the terminal tubes. Pneumonia is always present; it may be slight or extensive, and in advanced cases the pleural cavity may contain an abundance of serous fluid. Emphysema is the rule. The hairworm may cause a diffuse pneumonia, and, according to Hall,[14] a "nodular or pseudotubercular pneumonia." That nodules in the lungs of swine produced by parasites and by miliary tuberculosis, in most cases, cannot be differentiated by the naked eye, has been reported by Day, Bengston and Raffensperger.[15] They identified parasitic nodules by placing them between two heavy glasses such as are used for the examination of pork trichinae. The parasitic embryos, as well as characteristic clusters of lymphocytes, are distinctly seen without staining at a magnification of 40 to 60 diameters. The nodules are 1 to 5 mm. in diameter and are chiefly located in the posterior lobes.

In experimental infection of swine, Schwartz and Alicata[13] observed that the most marked pathological changes were petechial hemorrhages visible on the pleural surface as early as three days after ingestion of larvae. These hemorrhages were caused by the penetration

of the walls of the capillaries as the larvae entered and accumulated in the alveoli; with the growth of the worms consolidation and emphysema developed. In mature sheep, subject to repeated migrations of larvae one may find emaciation, edema and foci of pus in the mesenteric lymph glands, edema of the abdominal viscera, and fluid in the peritoneal cavity.

Symptoms.—In *sheep* the onset is gradual. They first show a soft low cough, followed in a few days by unthriftiness, poor appetite, and anemia. As the respiratory symptoms progress the sheep stands with a lowered and extended head. There are rapid breathing, a tenacious dirty mucopurulent nasal discharge, easily induced cough, and distinct râles on auscultation over the lungs. Diarrhea is common. The disease may be suspected whenever a number of animals in a group are affected. Pneumonia is usually present and the mortality is high. While it is generally stated that the disease is bronchitis, one must consider that in every bronchitis there is some pneumonia and that it may be extensive. According to Freeborn and Stewart,[6] "Animals with adequate food and shelter frequently harbor many lungworms, but they seldom show any symptoms of this parasitism until their general resistance is lowered by other causes."

Calves are among the most susceptible animals and they suffer severely. The first to be attacked may succumb to an acute diffuse pneumonia within two or three days of the onset. Coughing becomes paroxysmal and frequent, and suffocation may threaten. When the attack is more prolonged one observes emaciation, anemia, debility, sunken eyes, rough coat, poor appetite, diarrhea, and sometimes hemoptysis. In this form the animal lives for from three to five months. This form begins with an occasional cough that is described by Smythe[3] as a "bark," accompanied by a sudden protrusion of the tongue. After the disease becomes well-established there is a tenacious nasal discharge that may be slight or abundant. In severe cases the cough becomes weaker, dyspnea increases, and the animal shows open-mouth breathing with lowered and extended head and protruded tongue. Pulmonary emphysema may become extreme and lead to subcutaneous emphysema with distinct crepitation of the skin on palpation.

A 2-year-old heifer affected with lungworm disease at pasture in July had been unthrifty for a month. There was open-mouth breathing with the tongue protruded and with occasional spells of coughing. On being forced to move, dyspnea and cough were intensified and the breathing was like that of a horse affected with an advanced case of heaves. Moist crepitating râles were present over the lower half of both lungs. Pulse 80, respirations 60, temperature 104.6° F. After the ap-

pearance of these symptoms, there was progressive weakness until death five days later.

The chief symptoms in *pigs* are cough, loss in condition, and stunted growth. Deaths in young pigs are occasional.

Diagnosis.—This is based on the number affected, the clinical symptoms, and the presence of larvae in the feces. Postmortem examination reveals bronchitis, pneumonia, emphysema of the lungs, and above all, worms in the bronchi. A rapid method for the diagnosis of verminous bronchitis in sheep has been described by Vajda:[16] "Where the feces are of normal consistency, individual pellets from each sample are placed on a microscope slide in three to five drops of water and are allowed to stand for 15 minutes or longer. The pellets are then removed by means of a pair of forceps and the perfectly clear underlying water is examined for larvae, which are principally found at the edges of the drop of water. It is important not to injure the fecal pellet as otherwise the water enters it and the larvae do not leave so readily. In heavily infected cases two or three seconds are sufficient for the larvae of the *Dictyocaulus* to show themselves in water." These may be seen with a hand lens.

Treatment.—Medication of the lungs for the removal or destruction of mature worms in the lungs is regarded by those having wide experience as of doubtful value. In many outbreaks, especially in calves, the invasion is light and the symptoms gradually recede. Hall[14] refers to experiments conducted by Gilruth in New Zealand, who treated three lots of lambs with drugs, and gave good food with no medication to a fourth; the treated lambs gave a mortality of 25 to 50 per cent, while the lambs in the untreated lot all improved rapidly in health and condition. Since the lung tissue is less resistant to irritants than the lungworms, and the innumerable ramifications of the bronchi make complete contact with them practically impossible, the value of injection into the trachea of disinfectants or irritants would seem to be doubtful. This is expressed by Hall[14] in the suggestion that the injection or inhalation of poisons to kill lungworms commonly injures the lungs, but rarely injures the lungworms. Experience shows that after an attack of lungworm disease in sheep has been active for about six weeks the survivors of the flock have developed an immunity which protects them against further loss.

McGrath[17] reports that the New South Wales departmental formula is safe, and apparently effective, in the treatment of lungworm disease in sheep. It consists of:

Ol. terebinthinae	1.0 cc.
Creosotum	0.5 cc.

Ol. olivae .... 2.0 cc.
Chloroform .... 0.5 cc.

This is mixed and injected intratracheally as a single dose; it may be repeated twice at intervals of from two to four days.

Calves have been treated with intratracheal injections of gasoline (3-5 cc. daily), but reports on the effect of this treatment are conflicting. Some veterinarians have reported sudden death from suffocation following the injection, while others have reported improvement. These variations are probably determined by the number of worms in the bronchi; when they are present in great numbers suffocation may result. Another remedy consists of turpentine and olive oil each 100 parts, creolin 10 parts (inject 5-10 cc. into the trachea). Repeat twice at four-day intervals. In 1916 Herms and Freeborn[18] recommended chloroform for lungworm disease in goats and calves, the maximum dosage being 11 cc. for calves and 3 cc. for goats. The animals to be treated are confined in a yard free from vegetation. "The chloroform is best administered by tipping back the animal's head and injecting the desired dose of chloroform with a small pipette. Half the dose is administered in each nostril. The action of the chloroform is enhanced by stopping the nostrils with the hand or cotton plugs for a few moments after injection. . . . Two hours after treatment a saline purge of Epsom or Glauber's salt should be given the animal. . . . The chloroform stupefies the worms and at the same time irritates the throat and trachea, thus causing a prolonged paroxysm of coughing during which the worms are coughed up and swallowed. . . . Treatment should be continued at intervals of three to five days." A similar remedy consists of 2 ounces turpentine and 14 ounces sulfuric ether. Pour a teaspoonful down each nostril for lungworm disease in calves; repeat every three to four days.

According to Lytle,[19] a 10 per cent mixture of beechwood creosote and glycerin (2 to 4 cc.), intratracheally, has been found quite effective for lungworm disease in sheep and calves.

Opinions on the value of intratracheal injections are conflicting. Whether or not it is a natural recovery, clinicians and owners have often observed improvement after intratracheal medication. No claim is made for the treatment when there is an extensive involvement of the lungs.

The use of a vermifuge to expel larvae from the intestinal tract is indicated (see treatment of stomach-worm disease and treatment of nodular disease in sheep).

In the prevention of lungworm disease caused by *Dictyocaulus* one needs to consider infection from animals that act as carriers, yet do not

themselves have the disease. Such individuals are adult cattle, sheep, goats, and deer. In temperate climates the larvae of *Dictyocaulus* which remain in the soil or water are destroyed each winter by frosts, and the fields are reinfected each spring from mature animals. The water and feed troughs, therefore, should be so constructed that fecal contamination is impossible. So far as possible, provide running water instead of pools and troughs. In countries where infection is heavy it is advised that young cattle be kept indoors until the dew has dried off and removed from the fields by late afternoon, and that old pastures be plowed. Isolate newly purchased additions. Prevention of lungworm disease in sheep may be accomplished by the use of feed racks constructed in such a manner that fecal contamination of the food is prevented. A practical and inexpensive type of feed rack has been described by Turner.[20] See stomach-worm disease of sheep and goats, p. 209, for description. Avoid infected ground, moist pastures, grazing with older sheep, and the use of sheep manure on land where crops for green feeding are raised.

In the control of lungworm disease in pigs, chief importance attaches to earthworms. These are common in permanent hog lots where the soil is moist and covered with humus or litter. Infected pigs should be moved to clean pens or to recently cultivated fields free from litter. Provide clean running water if possible. Ingestion of earthworms may be lessened by abundant feeding and the use of nose-rings.

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PLEURITIS

Etiology.—(*a*) Acute primary pleuritis is infrequent. In the horse and cow it may result from exposure to cold and fatigue, especially in the cool months of the spring. The acute infections, such as influenza in the horse and hemorrhagic septicemia in bovines, may localize chiefly in the pleura, but such instances are rare and the diagnosis is uncertain unless the animal comes to autopsy. It is probable that in all cases there is an active infection. Acute diffuse pleuritis has been observed in a 4-month-old calf in excellent condition and with no previous history of disease; the cause was not determined.

(*b*) Acute secondary pleuritis is frequent. It occurs often in association with pneumonia, as in influenza, contagious equine pneumonia, hemorrhagic septicemia in cattle, enzootic pneumonia in calves, inhalation pneumonia, and advanced forms of lungworm disease. In acute septicemias, such as calf scours and hog cholera, there may be localization in the pleura; usually this is circumscribed and not clinically prominent, but pleuritis may be the chief lesion. Gross infection of the pleura of foals with strongyle larvae has led to acute serofibrinous pleuritis. Other causes are perforative wounds or severe contusion of

the thoracic wall, rupture of the esophagus, and extension from peritonitis or pericarditis.

(*c*) Chronic pleuritis is a frequent secondary form; it occurs in tuberculosis, necrobacillosis of the liver, pulmonary abscess, traumatic gastritis, and malignant tumors in the thorax. In our clinic, traumatic gastritis exceeds all other causes combined.

Morbid Anatomy.—The postmortem changes depend upon the cause. In acute infections one finds extensive hemorrhages of the serosa. In cases that are slightly less acute there is a serofibrinous or purulent exudate and often slight or extensive adhesions. In verminous pleuritis and tumor formation there is an abundant serous exudate. Traumatic gastritis is characterized by extensive adhesions and abscess formation involving both the lungs and the pleura.

Symptoms.—*Acute primary pleuritis* has a sudden onset. Usually the animal is found sick in the morning of the day following exposure. One finds anorexia, depression, congested mucosae in the horse, an increase in the pulse, rapid shallow breathing with an expiratory lift, and a moderate fever. In the early stages there may be colic. A suppressed cough may be present and induced, or there may be no cough. On auscultation one hears a weak vesicular murmur and friction sounds. Percussion and slight pressure with the thumb in the intercostal spaces cause pain. The course is brief and improvement is marked within twelve to twenty-four hours. It is possible that some of these are cases of pleurodynia (intercostal pain) instead of pleurisy.

Acute secondary pleuritis may not be recognized except on autopsy. But in association with equine pneumonia it presents definite and characteristic symptoms. When it develops at the onset of the attack there are severe chills, distress, and a high fever. When it appears later as a complication, the chest cavity gradually fills with fluid, expiratory dyspnea increases from day to day, and percussion reveals an area of distinct dullness over the lower third or half of the chest wall. This area of dullness is marked above by a straight horizontal line, the percussion sound passing abruptly and distinctly from dullness to resonance. Exudation of a large quantity of fluid may also occur in tumor formation, in verminous pleuritis, and in hemorrhagic septicemia in cows. In all cases of acute secondary pleurisy the prognosis is grave, but in acute infections recovery may occur if the exudate is relatively free from infection. In the horse, pleuritic adhesions may form and leave the animal with symptoms of heaves.

Chronic pleuritis occurs most often in association with traumatic gastritis in cows, and it is described under the general subject of traumatic

gastritis. In general any well-marked chronic pleuritis is associated with chronic bronchopneumonia. The identifying symptoms are pain and dullness on percussion and friction sounds on auscultation.

Treatment.—At the onset of an attack one may alternate hot and cold packs on the chest or apply spirits of mustard (5 to 10 per cent) or a mild mustard paste. For relief of pain, salicylate of soda 2 to 3 ounces (30-90 Gm.) is useful. While laxatives and diuretics are commonly recommended, the value of either as a routine is questionable. If there is a distressing cough, one may prescribe for a horse the combination of ammonia and belladonna recommended in coryza and bronchitis. When serous exudate accumulates in the pleural cavity it should be removed early through a canula inserted at about the seventh intercostal space. If necessary this operation may be repeated daily. Do not wait until a large accumulation of fluid causes expiratory dyspnea and other distressing symptoms; remove the fluid as soon as its presence is recognized. If the fluid is turbid or flocculent it suggests an unfavorable prognosis. The injection of adrenalin (10 to 15 cc.) through the canula after removal of the fluid is said to be beneficial. The use of air in a similar manner is recommended in human medicine. In acute primary pleuritis antibiotics are indicated.

DISEASES OF THE DIGESTIVE SYSTEM

CATARRHAL STOMATITIS

This is mild acute inflammation of the oral mucosa characterized by redness, swelling, and salivation. Inflammation of the gums is termed *gingivitis,* of the tongue, *glossitis.*

Etiology.—*Traumatic injuries* are a frequent cause. Foremost in the horse are sharp, loose, or split teeth. Next in importance are vegetable awns from barley, barnyard grass, and foxtail. Less frequent are injuries from harsh bits, and rough handling with a rope or cord in the mouth. *Chemical irritation* results from eating decomposed food, such as garbage, and from the administration of caustic drugs like chloral or ammonia. Prolonged use of mercury, lead, iodides, and arsenic may induce stomatitis, but this form rarely occurs in domestic animals. It is possible that stomatitis caused by eating white clover, alfalfa, and other irritant forage is chemical in origin. According to Fröhner, uromyces, and red and black rusts that appear on clover may cause various kinds of inflammation of the mouth. Catarrhal stomatitis may result from *extension of infection* from adjacent structures—from an abscess in the submaxillary region, empyema of the sinuses, pharyngitis, strangles, abscesses in the masseter muscles, etc. In *gastrointestinal* catarrh stomatitis occurs partly as a manifestation of the disease, partly because of decomposition of food and saliva in the mouth.

Symptoms.—Acrids and corrosives cause diffuse swelling and redness of the mucosa, salivation, and partial or complete refusal of food. Wound infections in the region of the tongue cause anorexia and edematous swelling in the intermaxillary space. After teeth abrasions the animal eats slowly, turns the head sidewise when chewing, or refuses food entirely. Horses tend to "drive on one line," and they may bite and chew the manger. With the aid of a speculum and flash light, examination will reveal various degrees of injury from slight recent abrasions to older and deeper ulcer-like defects. These are usually found on the margins of the tongue, near the lower border, or on the cheeks. Stomatitis from awns develops on the mucous membrane of the lips, and the inflammation may extend to the skin. The lesion is circular or irregular in outline. The surface is rough and dirty-yellow from the accumulation of awns and inflammatory debris. This thickened circumscribed area is surrounded by an inflammatory zone. Affected horses eat slowly and are unthrifty.

Treatment.—Remove tooth projections and splinters, and curette away awns. Apply antiseptics, such as potassium chlorate (4 per cent), alum (3 per cent), or permanganate of potash (2 per cent). After removal of awns the affected parts may be touched with lunar caustic and swabbed daily with a 10 per cent solution of silver nitrate.

VESICULAR STOMATITIS

Definition.—Vesicular stomatitis is a superficial inflammation characterized by the development of thin-walled vesicles or blisters containing clear, or yellowish serous fluid, not due to the virus of foot-and-mouth disease. These rupture early, so that the usual mark of identification is a superficial erosion partly covered around the margin with a thin narrow fringe of whitish membrane. In veterinary literature the terms vesicular and aphthous are sometimes applied to identical conditions. Probably this is a result of the similarity of the symptoms after the distinctive lesions have passed. Vesicular stomatitis is symptomatic of foot-and-mouth disease, contagious vesicular stomatitis in horses, cattle and swine, and vesicular exanthema in swine. Aside from the contagious forms, vesicular stomatitis is apparently infrequent in the United States. It is said to be occasional and mildly enzootic among pastured horses in the Mississippi Valley, and it is commonly attributed to acrid substances found on clover, rape, and similar roughage. In the author's experience, forms of stomatitis that might have passed for vesicular have been of the aphthous type. Of the numerous names employed, especially in the European literature, to describe limited enzootics of unknown cause, the term aphthous seems to include the majority.

APHTHOUS STOMATITIS

(Mycotic; Ulcerative; Erosive; Papulous; Follicular; Sore Mouth of Cattle; Thrush; Pseudo-Foot-and-Mouth Disease)

Definition.—Aphthous stomatitis begins with the formation of circumscribed, yellowish necrotic spots, 2 to 3 mm. in diameter (*aphtha—little ulcer*). The necrotic tissue soon sloughs, leaving small circumscribed ulcers that quickly heal (discrete), or that fuse and finally lead to secondary gastrointestinal or other complications. The feet, udder, and other parts of the body are sometimes involved.

Etiology.—It chiefly attacks pastured cattle in the fall, but it also affects stable animals turned in the yard daily. The disease may be sporadic or slightly enzootic. It is frequently reported in the United

States, Canada, and Europe. The cause is unknown. Mohler[1] has termed it mycotic on the theory that it is caused by fungi on the roughage. Since its occurrence is chiefly among cattle pastured or fed on fresh legumes—alfalfa, white clover, sweet clover—this theory seems logical but it has not yet been proved. Fröhner[2] refers to a report by Berndt, who described a fatal stomatitis and rhinitis in lambs due to the fungus *Polydesmus excitosus;* the condition resembled foot-and-mouth disease. Clover disease affects horses pastured on luxuriant alsike or sweet clover. Fröhner[2] attributes it to the fungus *Uromyces apiculatis.* Clover

Fig. 5.—Aphthous stomatitis, severe.

disease in horses has been observed and reported verbally in the United States. *Polydesmus excitosus* is reported in America upon all members of the rape family—mustard, cabbage, turnip, etc., while more than 2,000 rusts have been described. It is probable that some members of this group are partly responsible for the various forms of stomatitis that result from grazing on certain plants, such as the clovers.

Udall[3] observed a number of cases of aphthous stomatitis in cattle, affecting the mouth in mild cases, and the udder, coronary region, and other parts of the body in severe types. One that came to autopsy developed the disease in an alfalfa pasture. The stomatitis was severe and ended fatally after about six weeks. In addition to the lesions on the teats, coronary region, skin of the back, and mouth, the esophagus was covered with superficial ulcers, and the gastro-intestinal tract was

extensively inflamed. Kantarowicz[4] has described a similar form in cows; it was attributed to feeding the first crop of green clover.

Infection seems to be a cause. Pusch[5] has described a form that appeared in a government breeding herd among bulls recently imported from Switzerland. Soon after unloading, affected animals developed slight salivation. The lesions were flat, the size of a pea and larger, on the hard palate, interdental space, lips, and muzzle. He named it *erosive stomatitis* due to infection in young animals whose resistance had been lowered by travel. It was non-transmissible. Ostertag and Bugge[6] have reported a similar infection in calves that was transmissible—*stomatitis papulosa specifica*.

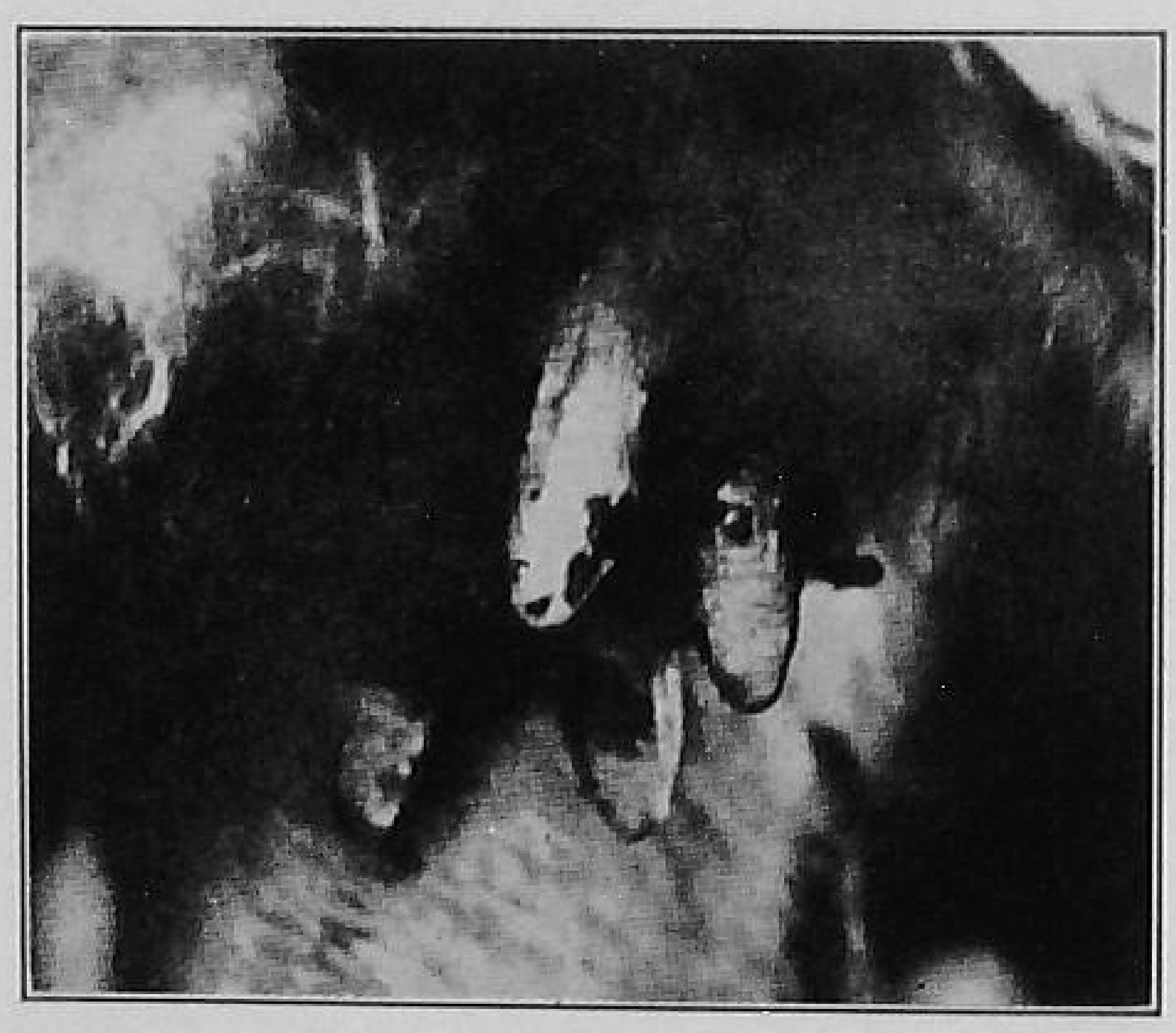

Fig. 6.—Teat lesions in sporadic aphthous stomatitis.

Ulcerative stomatitis is symptomatic of foot-and-mouth disease following rupture of the vesicles, rinderpest, clover disease in horses, various septicemic conditions of unknown cause, and hyperkeratosis in cattle; it has also been observed in malignant head catarrh. An outbreak of ulcerative stomatitis in cattle in South Africa, described by Keppel and Robinson,[7] was marked by stiffness, salivation, ulcers on the dental pad, gums, tongue, and cheeks, swelling of the neck, and a low grade fever. Stomatitis was not always present and the cause was not determined.

Symptoms.—In *mild cases* small superficial erosions on the mucosa of the lips are the only symptoms. In general, the prognosis is good and the mortality low. Mohler[1] writes that in the serious outbreaks investigated the mortality has been less than 0.5 per cent.

In the more *severe* types yellowish papules appear on the muzzle,

eating stops, and saliva drools from the mouth. In a short time sloughing and fusion occur, when the muzzle becomes yellowish, moist and necrotic. In the mouth erosions may be found anywhere—gums, cheeks, hard palate—and the odor is fetid. Fissures at the coronary region cause lameness, papules may form along the back, and superficial ulcers may form on the teats. Loss of condition is rapid and the pulse is fast, but the temperature tends to remain normal. Finally, enteritis with diarrhea sets in and the animal dies.

Treatment.—Change the food, either by removal from pasture, or by substitution of other roughage. Treat the mouth as in other forms of stomatitis. Fissures and ulcers around the feet may be covered with an alcoholic sublimate pack. Zinc oxide and sulfathiazole ointments are useful for the teat lesions.

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CLOVER DISEASE

Clover disease is described by Fröhner[1] under food rashes, by Marek[2] under the diseases of the skin. It is characterized by depression, nervous symptoms, dermatitis, and a severe stomatitis. It occurs in horses pastured on alsike clover (*Trifolium hybridum*) but the essential cause is unknown. A few cases have been reported in this country. It is ushered in by dullness, lowered head, and aphthous lesions of the tongue or other parts of the mouth. Frequently the white parts of the skin are affected with superficial necrosis, and in some cases the lesions are limited to the skin. At other times the effect is chiefly general, when the animal shows colic, bloody diarrhea, marked jaundice, hematuria, dizziness, paralysis, and amaurosis. A similar disease, with necrosis of the white spots of the skin, depression, rapid loss of condition, diminished milk flow, but without stomatitis, has been observed in our ambulatory clinic in stabled cows fed heavily upon alfalfa. On account of the wide variation in symptomatology and severity, a diagnosis

based upon the cause may be difficult. *Treatment* consists in withholding the irritating clover and treatment of the local symptoms.

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PHLEGMONOUS STOMATITIS

Definition.—A deep inflammation of the mouth, affecting chiefly the tongue, characterized by a serous or purulent infiltration of the connective tissue, and swelling. It is relatively infrequent.

Etiology.—Wounds of the tongue from barbed wire and similar objects, extension of infection from abscesses (strangles) in the intermaxillary or masseter muscles, severe forms of necrotic stomatitis or clover disease, and deep burning from acrid chemicals (ammonia, chloral, acids). It may be a partial condition in rinderpest, purpura, malignant head catarrh, and anthrax (gloss anthrax).

Symptoms.—The development is rapid and painful. On examination one finds drooling, separation of the lips by a protruded swollen tongue, a reddish or bluish swollen shiny mucosa, and an abundance of saliva and mucus. Fetor accompanies purulent infiltration or abscess formation. When due to injuries and caustics, recovery usually occurs in a short time; when due to infection, the course and termination depend on that of the primary disease. *Treatment* is the same as for other forms of stomatitis.

NECROTIC STOMATITIS

(Calf Diphtheria; Gangrenous Stomatitis; Sore Mouth in Pigs)

Definition.—Necrotic stomatitis is an acute inflammation, due to *Actinomyces necrophorus* (necrosis bacillus), characterized by caseous necrosis with the formation of ulcers, and toxemia. Occasionally the necrosis is dry, with no ulcers.

Etiology.—It is common in America in calves and pigs, in either a sporadic or an enzootic form. Severe losses in South Dakota, Wyoming, and Colorado have been reported by Newsom,[1] Melvin and Mohler,[2] and Elder.[3] Sucklings are chiefly affected, but in severe outbreaks mature animals may be attacked. Since a filterable virus has been found to be the cause of sore mouth (contagious ecthyma) in lambs, the necrotic lesions in this species are regarded as secondary *A. necrophorus* infections. In the eastern part of the United States necrotic stomatitis in calves is common even where the sanitation is

excellent. There is no strict seasonal occurrence of calf diphtheria in New York State, though it is more frequent in the winter. In the West it is reported not to occur in the summer months.

Actinomyces necrophorus is an anaerobe obtained in the form of filaments in smears made from the live tissue at the margin of the ulcer or other necrotic lesion. Stain with carbolfuchsin. Its habitat is manure, soil, the healthy intestine of swine, and possibly of other animals. Observations reported by Tunnicliff[4] indicate "that this organism normally remains alive under natural soil conditions for only a short time, although, exceptionally, a small percentage of the organisms

Fig. 7.—Necrotic stomatitis; tongue of a cow.

may survive for an extended period of time. It cannot be classed as a saprophyte of the soil." Most writers refer to it as a secondary invader that attacks only weakened tissue; that will not develop in a normal mucous membrane; and that enters through a slight break in the mucosa. Accepted predisposing causes are injuries to the mucosa from

sharp objects or stubble and exposure to filth. Yet the disease often occurs in the apparent absence of such causes, and the wide range of attack in many different organs under widely varying conditions suggests that primary infections are probable.

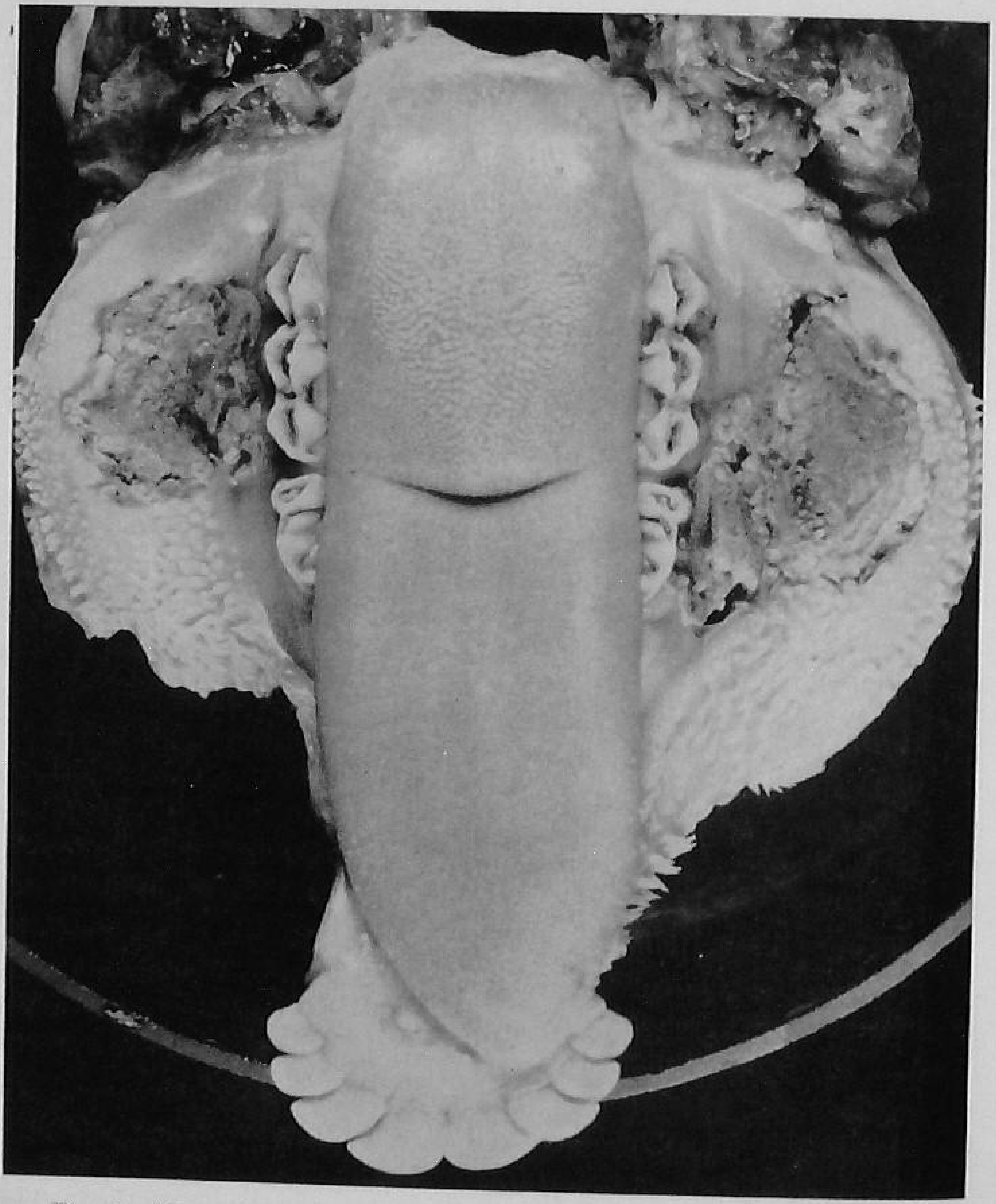

Fig. 8.—Necrotic stomatitis, calf. (Courtesy of James A. Henderson.)

Pathology.—The favorite locations of the characteristic necrotic ulcers are on the cheeks adjacent to the molar teeth and at the base or sides of the tongue. The base of the ulcer is a mass of yellowish, greenish, or brownish necrotic fetid material firmly attached to the underlying tissue. The offensive odor has been likened to that of

Limburger cheese. The lesion has been described by Mohler[2] as "a coagulation necrosis with subsequent caseation, and marked by a progressive invasion of the surrounding (deeper) tissue and a remarkable tendency to metastasis." The margin of the ulcer consists of a thick firm wall of granulation tissue. In severe outbreaks necrotic lesions may be found in the larynx, pharynx, trachea, lungs, esophagus, rumen, omasum, abomasum, and the skin, especially that of the lower extremi-

Fig. 9—Necrotic stomatitis. (Courtesy of James A. Henderson.)

ties and interdigital space; in these types there is advanced emaciation. This severe form in lambs has been described by Newsom and Cross,[5] and in calves by Elder.[3] A venereal form associated with sore mouth in sheep has been described by Melvin and others.

Symptoms.—The incubation period is from three to five days. The young may be affected within a week from birth. In calves there are depression, emaciation, anorexia, salivation, sucking of the tongue, swallowing movements, and a swelling of the neck or cheek. The most common location of the swelling is on the cheek. It is round, from 1 to 3 inches in diameter, and with rare exceptions there is an underlying ulcer that may be recognized on examination of the mucous membrane near the base of the molar teeth. Usually the temperature is

high. At first the lesions resemble small papules covered with scabs; these coalesce to form a necrotic area surrounded by a reddened zone. Necrosis may nearly sever the tongue, or the tissue of the cheek bordering the gums may be deeply sloughed. Pneumonia or gastroenteritis develop in prolonged attacks. In pigs the necrosis usually appears around the front teeth and tusks. Newsom[1] has described widespread fatal enzootics affecting swine of all ages. The necrosis loosened the teeth and invaded the bones of the face; large cheesy ulcers were found in the stomach and throughout the large intestines; similar ulcers developed on the skin of the legs, and in the sheaths of the males. In older animals the disease is less severe. In cows fatal enzootics of necrotic stomatitis, in conjunction with necrosis of the teats and the coronary region, may lead one to suspect foot-and-mouth disease. Instead of large vesicles and superficial ulcer-like defects, one finds a dry circumscribed necrosis that involves the entire thickness of the tongue, or the total diameter of the teat. The affected parts are brown and dry and the papillae of the tongue are lost. In the differential diagnosis one needs to consider hyperkeratosis.

Prognosis.—Calves and pigs that receive individual treatment usually recover in about two weeks. Outbreaks in any species may be mild, but where filth or underfeeding prevail, the mortality is high.

Treatment.—Remove the necrotic tissue and touch the affected parts with lunar caustic, or swab the surface with a 10 per cent solution of silver nitrate. Other useful disinfectants are Lugol's solution of iodine, or 4 per cent boric acid. Elder[3] reports that in Wyoming "we have found the best treatment to be a 10 per cent solution of potassium permanganate to which 1 per cent active chlorine has been added. . . . Dakin's solution may be used after adding 10 per cent by weight of potassium permanganate crystals." The administration of sulfapyridine (3 Gm. per 100 lbs. body weight daily) has proved to be especially beneficial in this disease, as well as in necrotic laryngitis.[6] Hayes and Wright[7] have reported excellent results in the treatment of an extensive outbreak of calf diphtheria in Montana with sulfamethazine; one 15-Gm. bolet (approximately ¾ to 1½ grains per lb. of body weight) once a day for two days; 2,785 cattle were treated in the course of two weeks. Similar results are claimed for sulfamerazine.[8]

Prophylaxis: Segregate the sick, cleanse and disinfect stables, and examine well animals daily for new cases.

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SALIVATION

(Ptyalism; Sialosis; Supersecretion)

Salivation is a symptom of various affections; often it is apparent rather than real, the saliva not being swallowed. Excessive secretion may be due to the following causes:

(1) *Local irritation* from stomatitis, defective teeth, foreign bodies in the mouth, pharyngitis, foot-and-mouth disease, etc. Horses pastured on sweet clover often salivate freely. It is a marked symptom in clover disease.

(2) *Reflex stimulation* may cause profuse salivation in choke, or in acute indigestion. It is associated with impaction of the abomasum.

(3) *Direct nerve stimulation* increases the salivary secretions in
rabies, cornstalk poisoning and other brain diseases, and in affections of
the peripheral nerves. A similar effect results from the action of mer-
cury, iodine, pilocarpine, and arecoline.

(4) *Paralysis of the pharynx* (pseudorabies, rabies) causes abundant drooling of saliva.

Treatment.—Temporary relief may be obtained from atropine.

INFLAMMATION OF THE SALIVARY GLANDS

(Parotitis)

There are three groups of salivary glands—parotid, submaxillary,
and sublingual. These are occasionally diseased, and in cows abscesses
due to nonspecific infection may be closely associated with them,
causing tuberculosis of the lymph glands to be suspected. The type of
inflammation is either parenchymatous or suppurative (abscess).

Etiology.—The parotid gland may be infected from the entrance
of awns through Stenson's duct, from the extension of an inflammation
from the duct, or from the circulation. Specific infections, such as tuber-
culosis, actinobacillosis, and strangles enter through the lymph stream.

Abscess formation results from blows, hooks, and penetrating injuries. In certain localities mild inflammations of the parotid gland are common in horses.

Symptoms.—One first notes a swelling that conforms to the outline of the gland, or of Stenson's duct. In strangles, multiple abscesses of the parotid gland sometimes develop, and may cause an edema that extends to the surrounding tissues, presses upon the pharynx, and causes inspiratory dyspnea. The edema may also extend to the head, eyelids, and ears. In the acute form the gland is swollen and painful. Mastication is slow and difficult, salivation abundant, and the odor of the mouth fetid. In a week to ten days abscesses may rupture and recovery follow. Stenson's duct may remain swollen for months in equines, and then suddenly enlarge and involve the parotid gland in a firm painful swelling.

Inflammation of the submaxillary salivary gland is observed in cows. It is not always clear that swellings of the gland are inflammatory in character; aspirated fluid may be straw-colored and contain epithelial cells and leucocytes, but no bacteria. The gland may be left permanently enlarged and firm.

Treatment.—In the acute, painful stage, cold applications afford relief. The development of edema suggests the formation of an abscess, and whenever pus is located it should be promptly released. When the gland tends to become indurated, resorption may be hastened by the use of tincture of iodine, or of direct injection into various parts of the gland of 5 cc. of Lugol's solution at intervals of a week to ten days.

PARALYSIS OF THE PHARYNX

(Glossopharyngeal Paralysis)

Etiology.—Paralysis of the pharynx is occasionally seen in equines. It is characterized by inability to swallow, absence of general symptoms, and absence of paralysis of other organs. All cases that have come under the observation of the writer have been sporadic—one of a pair, or a single driver. The paralysis is usually complete without recognizable cause. Formerly such cases were often termed "spinal meningitis" on the assumption that the lesion was in the spinal meninges. More recently the term "forage poisoning" (botulism) has been applied on the theory that toxic substances in the food cause the paralysis. In botulism, paralysis of the pharynx exists in conjunction with extensive peripheral paralysis; in meningitis, paralysis of the throat is usually absent. Following an attack of strangles, paralysis of the throat may have a good prognosis. Taylor[1] writes that "the symptoms in pharyngeal paralysis are characteristic of lesions of

the nuclei of origin as seen in bulbar paralysis." According to Law,[2] "paralysis usually implies disease of the bulb at the roots of the vagus and glossopharyngeal nerves, or swelling affecting these nerves or the sympathetic along its course." Bulbar paralysis, "paralysis of the pharynx," is one of the symptoms of rabies, other forms of encephalitis, abscess of the brain, and intoxication (botulism). Post-strangles paralysis is probably caused by delayed resorption of retropharyngeal abscesses.

Symptoms.—In the few cases that have been seen by the writer, the horse has been apparently normal at night and in the morning has not been able to swallow. In two cases the animals had been at regular work, were turned to pasture for one or two days, and were found in this condition. Rarely there is a history of a "cold" followed by a progressive paralysis that finally becomes complete. Repeated examinations fail to reveal any other symptom except inability to swallow. This is manifested by a constant bilateral grayish white nasal discharge that changes in color according to the nature of the food; in pastured horses it is green. There may be a cough. Finally, the general condition becomes poor, and unless entrance of foreign material into the trachea causes earlier death from inhalation pneumonia, the horse dies of exhaustion. A transient paralysis of the pharynx is occasionally observed in cows.

Treatment.—A few recovered cases have been recorded, but it is not clear that the course was modified by treatment. The writer treated one case with local application of electricity for a month, the horse being nourished with gruel given through a stomach tube. Intervals of slight improvement, manifested by decreased nasal discharge and less coughing, were brief, and death was the final result. The use of vitamine B_1 (thiamine hydrochloride) in doses of 0.5 Gm. per day intravenously has been suggested because of recovery following its administration to two or three horses. This is given with dried brewer's yeast, 500 Gm. daily in water through a stomach tube.

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CHOKE

(Obstruction of the Esophagus)

Etiology.—*Cattle* are often choked because it is their nature to swallow without previous thorough mastication. Many kinds of foreign bodies, as well as large pieces of roots and other food, reach the stomach

unchanged. Choke in bovines is most frequent when certain fruits or vegetables, such as cabbages, apples, and roots are abundant; it is especially common after storms and when apples and roots are wet. Ninety per cent of the choke in cows treated in the ambulatory clinic at Cornell University has been on apples. Other objects are potatoes, beets, turnips, ears of corn, and cabbage stumps. Less frequently cows are choked upon metallic substances, such as pieces of tin and glass. Smith[1] has described a case of choke in a cow caused by swallowing a table fork; recovery followed operative removal from the lower third of the cervical region. As a rule large objects lodge in the cervical part of the esophagus, but occasionally they are found in the thoracic portion. Shigley[2] has reported the finding of an entire beet partially protruding from the esophagus into the reticulum. Recovery followed rumenotomy and removal of the beet with the aid of a wire hook.

Horses choke on ears of corn, roots, or hay, but more often from eating greedily of dry food, such as oats or bran. In equines it usually occurs in animals ten or more years of age. Habitual choke in horses may be due to chronic circumscribed esophagitis that has resulted in stricture, or to improper mastication in old animals with poor teeth. Choke may follow narcosis, or a loss of consciousness from disease. In such cases the esophagus remains partially paralyzed and dry hay or grain is apt to lodge. Capsules administered to horses suffering from colic sometimes lodge and dissolve in the esophagus where their contents induce inflammation. Danger from a broken capsule is largely prevented by first giving a small dose of arecoline ¼ grain (0.015 Gm.).

Symptoms.—*Cervical choke in cows* is first recognized by salivation and bloating; the latter may cause anxiety and restlessness. Coughing, chewing movements, retching, and forced swallowing movements occur. The patient makes no attempt to eat or drink. Palpation of the esophagus in the jugular groove usually reveals an apple or root in the upper part of the esophagus; often it is located immediately behind the pharynx.

Thoracic choke in cows may cause marked tympany with the resultant alarming symptoms of restlessness, grunting, delirium, extreme dyspnea with open mouth, salivation, and gasping. After the tympany has been relieved through a canula in the rumen the acute distress vanishes. The animal may eat and drink, but the ingesta will be regurgitated with retching and cough. On passing a probang or stomach tube, the obstruction is usually located near the termination of the esophagus, less frequently near the anterior end of the thorax. In rare cases choke is followed in one or two days by subcutaneous emphysema. This may be a partial symptom of interstitial pulmonary emphysema

induced by retching; less often it is due to perforation of the esophagus.

Metallic substances tend to lodge in the pharynx or upper part of the esophagus where they cause severe inflammation. If the esophagus is perforated there develops a circumscribed edematous crepitating swelling. As eructation occurs the emphysema increases. Within two or three days the temperature and pulse are elevated, and the nostrils are partially filled with dry regurgitated food. Complete anorexia for food and water is present from the beginning. Since the obstruction is incomplete, tympany does not at first develop, but later the swollen tissue obstructs the esophagus and bloating gradually appears. Prompt relief is afforded by means of an operation. But when infection has advanced too far, death occurs in about a week.

In *cervical choke in horses,* a swelling is both seen and felt at about the middle of the neck. In habitual choke in old horses suffering from chronic esophagitis with stricture and dilatation, eating may be slow. When food lodges in the thoracic portion, the head and neck are extended, the upper lip is raised, and undulating waves pass up the jugular groove; this may be followed by cough. In some the tendency to choke persists until finally, after an attack of strangling, the thoracic portion of the esophagus fills with food for a considerable distance and is distended to two or more inches in diameter; this distension is carried forward and up the cervical region where it may be seen as a well-defined swelling. On palpation, deep crepitation of the mass within the esophagus may be felt. Such cases are usually fatal; in addition to choke, inhalation pneumonia develops.

In younger animals, after greedily bolting oats or bran, the horse is first observed to strangle. Eating stops, the nose is drawn towards the chest while the neck curves upward, and severe spasmodic contractions of the esophagus develop; this retching may cause the horse to squeal with pain. Salivation is abundant. Restlessness, colic-like movements, cough, and regurgitation of food and saliva through the nostrils are observed in the early stages. Massage of the mass in the esophagus may induce painful retching. These symptoms are most marked at first; after an hour or two the intervals between attacks of retching are longer and the attacks are less severe, the patient becomes quiet, and may even return to eating.

While the early symptoms are often alarming to the owner, the probability of immediate death from choke is remote. If unrelieved, choke may lead to a fatal paralysis and pressure necrosis of the esophagus, or death from inhalation pneumonia. Neither of these conditions can be avoided by hasty and ill-managed treatment, which often kills animals that might have recovered had they been left alone. Choke on

apples in cows is relatively mild; the chief immediate danger is from acute tympany, which may become alarming if the obstruction is complete. In most cases choke is relieved spontaneously within twenty-four hours, but it may persist for two or three days, or even a week. When the body is large and irregular and is lodged in the thorax it may become wedged tightly into the esophagus, gradually cause swelling, and thus create progressive difficulty to spontaneous or operative removal. When obstruction of the esophagus is complete, cows may require almost constant attention to relieve the tympany; and whenever tympany is absent or slight in choked cows, it indicates that the esophagus is not entirely closed and that the danger from fatal complications is comparatively slight. When the cow is choked on large hard roots, like potatoes, beets, or turnips, fed without cutting, the case is more serious and if possible the object should be dislodged; one can depend less on expectant treatment.

Choke in horses, likewise, is not apt to result in immediate death; spontaneous recovery after a few hours to two or three days is the rule. The mass gradually moistens and softens, a change of great value in conjunction with proper treatment.

Diagnosis.—Cows affected with bronchitis, even in a mild form, are sometimes suspected of being choked; this is due to the lowered position of the head, the cough, the open mouth, and the protruded tongue. Acute bloat, for similar reasons, has been mistaken for choke, even to the extent of passing a probang to dislodge the supposed body, and choke has been diagnosed as indigestion. Choke on metallic substances lodged in the pharynx or upper part of the esophagus has been diagnosed as pharyngitis.

Treatment.—Tympany in cattle may be controlled by tying a wooden gag, or rope, in the mouth. If this does not afford relief, trocarize the rumen. It may be necessary to keep the canula in position for hours, holding it with sutures. Mechanical assistance usually affords prompt relief to cattle choked upon hard pieces of food. When an apple or similar body is located in the cervical region, an assistant should force it to the pharynx by placing a hand on either side of the throat and pressing upward with both thumbs against the body. It can be held in position in the same manner until the operator passes his hand through the pharynx and grasps it in the upper end of the esophagus; in the great majority of cases this method is successful. Some report success with a wire bent double to form a loop; the loop is passed behind the object which is then removed by traction. Or a No. 9 wire 30 inches long with a blunt ½-inch hook bent at the end is firmly fastened in the foreign object with the fingers and an assistant pulls on the wire.

With a method described by Hall,[3] an obstetrical chain is placed firmly around the neck posterior to the foreign body in the cervical region, and pig forceps to which elongated handles have been welded are passed to the obstruction, opened somewhat and attached to the object. To control violent movements or intense spasmodic contractions of the esophagus, it may be helpful to administer a narcotic in the vein.

In both methods a mouth speculum is indispensable. A probang is essential to the prompt relief of thoracic choke, and in cows it may be useful in cervical choke. Instead of a specially constructed probang, one may use an ordinary stomach tube provided with a stilette—a tube alone is too flexible—or an old smooth piece of ½- to ¾-inch rubber hose. With the aid of one or two assistants and a mouth speculum the passage of a probang in cows is not difficult. The surface of the tube should be well oiled, and the operator must exercise care in the amount of pressure to be exerted; repeated attempts at intervals of a few hours or days may finally result in dislodgement. The danger from a probang consists in injuries to the esophagus from rough manipulation; or too great pressure against the object may deflect the end of the tube against the tissues.

In horses a dry mass of ground feed is less readily dislodged by a probang, and one must use more caution to avoid injury. The ordinary stomach tube with a stilette is perhaps the safest form of probang. The use of a tube to convey water against the occluding mass is a dangerous practice. As sometimes used, it consists in pumping water into the esophagus and quickly syphoning it back. Sometimes the esophagus is exposed for 4 to 6 inches; it is then ligated with a 2-inch bandage to prevent regurgitation, and water under slight pressure is injected into the tube. To control pressure the fingers should be kept on the esophagus below the point of ligation. Such an operation is indicated only when one with experience and judgment decides that the usual methods of relief will fail.

Arecoline has come into use in the past few years in the treatment of all forms of choke. It increases peristaltic action, while the increased secretions that follow its administration probably tend to soften the mass and lubricate the mucosa. The dose is ½ to 1 grain (0.03 to 0.06 Gm.) repeated once or twice daily, often in conjunction with strychnine sulfate ¼ grain (0.015 Gm.). In no case should the patient have access to food or water until the obstruction has passed. In most cases of choke in the horse, this precaution alone will lead to spontaneous relief within twenty-four hours. Apomorphine hydrochloride ½ to ¾ grain (0.03 to 0.045 Gm.) is reported by Bradley[4] to have repeatedly given prompt relief. Chloroform narcosis in both horses and cattle has been

employed on the theory that it relieved "esophageal spasm" and rendered the probang more effective. Williams[5] has described a case of "hay choke" in a horse that recovered spontaneously on the sixth day after repeated failure to pass the probang under complete anesthesia.

A method that occasionally is effective consists in forcing animals to jump ditches or obstructions or exercise violently for a few minutes. Occasionally the choke is suddenly relieved when the victim falls from suffocation. A few cases have been described where cervical choke on a dry bolus of provender has been softened and broken down by passing a needle through the skin into the mass and injecting into it oil or water. In a similar manner, cervical choke on a beet has been relieved by passing a trocar into the mass and breaking it down mechanically.

For relief of thoracic choke in the horse, caused by beet pulp, Doenecke[6] has passed two stomach tubes, one through each nostril; water is pumped through one, while the dislodged material flows back through the other. He also gives atropine sulfate ½ grain (0.03 Gm.) to relax the spasms and diminish the flow of saliva. For choke that does not respond to usual methods of treatment before the third day, Krüger[7] has passed a stomach tube, connected it with a water faucet and given the force of from 1 to 1.5 atmospheric pressure.

For the immediate relief of low choke in the horse, Ferguson[8] recommends that after passing a stomach tube warm water containing a little soda be injected and syphoned back repeatedly; the head is kept down either by tying it close to the floor or by standing the horse on a steep incline. To overcome resistance by the horse he administers chloral hydrate (300 to 350 cc. of a 12 per cent solution) intravenously, and to control distress atropine sulfate, ¼ to ½ grain (0.015 to 0.03 Gm.). Cozart[9] prescribes 2 grains (0.12 Gm.) atropine sulfate for choke in a horse or cow.

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SPASM OF THE ESOPHAGUS

(Esophagismus; Cardiospasm)

Spasm of the esophagus, commonly described as a rare neurosis, is of doubtful occurrence as a functional nervous disorder in animals.

Etiology.—Bolton[1] has described an attack of esophageal spasm in a nine-months-old colt affected with strongyles and ascarids; the symptoms disappeared after about six weeks. A transient esophagismus following sulfonal anesthesia in a horse has been described by Berton.[2]

Spasm of the esophagus is usually a symptom of some organic lesion, especially esophagitis; less frequently it is due to foreign bodies, larvae of bot-flies, contusion, wounds, ulcers, etc. It may have a central origin in rabies or tetanus. Ferguson states that it occurs most often in foals when they start to swallow food other than liquid.

Symptoms.—Clonic spasms of the cervical muscles and the esophagus are the dominant symptoms. Regurgitation of food and water is a part of the history, but this may not be constant. Passage of a stomach tube will reveal the presence of an obstructive lesion or foreign body. If no obstruction is met, and the spasms are confined to the cervical region, one may suspect a lesion of the mucosa—ulcer, fissure, or parasite. If the onset is of recent origin one may learn that the animal has recently been treated for colic; this suggests injury from a corrosive drug. When the condition has prevailed for months, and the date of the onset is unknown, an anatomical diagnosis cannot be made on the mere presence of spasms. Bolton has written the following description of his case: "Cough frequent and painful. Bilateral mucopurulent nasal discharge. On drinking water he was suddenly seized with an attack in which he lowered the head, became very restless and attempted to vomit, giving a low moist cough and discharging through the mouth a quantity of water and saliva. He began at once to make frequent swallowing movements, continuing until the esophagus became filled to the pharynx with saliva. Then he lowered the head, attempted to vomit and expelled the saliva through the nostrils and mouth again. The attack lasted for half an hour or more, when suddenly the frequent swallowing movements ceased and the colt became quiet and drank water. During one of the attacks the stomach tube was passed without difficulty, but after withdrawal the operation could not be repeated. Recovery was complete. The attacks were irregular and had no relation to eating or drinking. Similar attacks were reported in a seven-months-

old full brother." Permanent recovery after passage of the tube is suggestive of cardiospasm.

Treatment.—Relief follows recovery from the basic disease. Symptomatic treatment consists in the administration of belladonna or other similar sedative. Passage of the stomach tube may possibly afford relief; this method is successful in man, and is highly recommended by Law, who advises that the end of the probang be smeared with solid extract of belladonna.

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STENOSIS OF THE ESOPHAGUS

With the exception of choke, obstruction of the esophagus is not frequent in animals. Obstruction stenosis is usually caused by chronic esophagitis, rarely by parasites or tumors. Compression stenosis from an enlarged tuberculous mediastinal lymph gland is not rare in cows. Compression from abscesses and tumors is rare.

Symptoms.—Compression of the esophagus in bovines induces symptoms not observed in equines; swallowing may occur normally but rumination is obstructed. The result is tympany that develops after eating. This gradually increases until chronic bloat seriously affects the general condition. Chronic tympany in cattle may also follow stenosis of the pylorus due to circumscribed tissue proliferation, as well as stenosis of the duodenum due to peritoneal adhesions and kinking of the gut; the latter may be found in traumatic gastritis. Stenosis occurring in esophagitis and choke are described under these subjects.

Treatment.—When due to a chronic pathological tissue change, treatment in large animals is not practical. Compression of the esophagus from acute inflammatory swellings may be relieved with cold packs.

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INFLAMMATION OF THE ESOPHAGUS

Acute Esophagitis.—Etiology.—Among the more frequent causes of esophagitis are: (*a*) Corrosive drugs, as formalin, chloral, and ammonia, administered in gelatin capsules that lodge and dissolve in the esophagus. (*b*) Choke from foreign bodies that cause injury by laceration or pressure—wire, glass. (*c*) Passage of a probang, whipstock, or broom-handle into the esophagus to dislodge a foreign body. (*d*) A partial condition in severe foot-and-mouth disease, rinderpest, ulcera-

tive stomatitis, malignant head catarrh, and necrobacillosis. Mild irritations cause catarrh. More severe ones cause croupous, diphtheritic, necrotic, or phlegmonous inflammations.

Symptoms.—In *horses* a mild form occurs after a capsule of chloral breaks in the esophagus. In certain cases of colic, capsules lodge apparently because of dryness of the mucosa and atony of the muscular walls. Shortly after the capsule is given, spasmodic contractions of the esophagus and cervical muscles occur at intervals of about ten minutes, gradually subside, and disappear in ten to twelve hours. In more severe forms the spasms may persist for two or three days; the animal is depressed and does not eat, while efforts to swallow water bring on spasms of the esophagus and painful regurgitation.

In *cattle* lacerations and contusions may cause various symptoms that persist for several weeks. The more severe form is nearly always the result of careless or hysterical use of the probang or its substitute, but it may result from choke upon a large irregular or sharp foreign body. Anorexia for food and water is observed, though water may be swallowed with more or less difficulty according to the degree of cough and retching thereby induced. At intervals there is a painful suppressed cough. Dullness and increased pulse are found. Subcutaneous emphysema in the jugular groove indicates perforation of the esophagus, and usually portends a fatal termination. More extensive lacerations often include injuries to the pharynx and larynx; the amount of injury that a cow may survive is remarkable.

In a cow the owner passed a broom-handle to dislodge an apple from the cervical portion of the esophagus. On the second day the apple was removed manually. On the third day the animal did not eat, and on drinking the water returned through the mouth and nose. There was a large firm swelling two-thirds of the distance down the neck, beyond which it was impossible to pass a stomach tube. In such a case slaughter may be indicated.

Treatment.—When there is no obstruction or perforation, chief reliance should be placed upon expectant treatment. Withhold food and water, though after two or three days water may be swallowed. As ability to swallow is regained, nourish with oatmeal water or with milk and eggs. Acute swellings in the throat, following the removal of sharp objects, may be relieved with steam inhalations combined with the application of cold packs. Direct application of drugs to the esophageal mucosa is of doubtful value; although silver nitrate, 1:5000 in water, as well as liquid petrolatum, are recommended.

Chronic Esophagitis.—This sometimes occurs in the thoracic region in horses where it is caused by injury from a probang or choke or by

corrosives that first induce an acute inflammation, leaving the mucosa partially denuded. As a result of the formation of granulation or scar tissue, a *stricture* may gradually develop. This leads to the obstruction of free passage of food until that part of the esophagus immediately above the constriction becomes permanently dilated—*esophageal dilatation*. The final effect is a "chronic choker." At last the esophagus becomes impacted with masses of food, paralysis is complete, and death from foreign body pneumonia follows in a week or ten days. The presence of an *ulcer* in the lower part of the gullet may result in spasm of the esophagus. In one of the writer's cases, a 10-year-old mare choked, salivated, and regurgitated on drinking cold water; this was the history for a year. On examination, attempts to induce choke by drinking were unsuccessful and there was no obstruction to the passage of the stomach tube. In such a case, one can only surmise the presence of an ulcer or some other circumscribed superficial lesion. Treatment is useless.

PARALYSIS OF THE ESOPHAGUS

Paralysis of the esophagus is symptomatic of affections of the medulla. According to Dexler, it results from affections involving the 9th to the 12th cranial nerves. It is observed in chronic esophagitis, when combined stenosis and dilatation prevent the passage of food. Following deep narcosis, paresis of the esophagus may remain for twenty-four hours and lead to symptoms of choke whenever an attempt is made to swallow solid food. It is often stated that in paralysis of the pharynx the esophagus is usually involved also. Ries[1] has described paralysis of the pharynx in which the horses were successfully fed through a tube fixed into a surgical opening in the esophagus; in the cases described it is evident that the esophagus was not involved. External pressure from tumors, abscesses, tuberculous lymph glands, etc., located in the jugular groove or thorax is a cause of paralysis. It is a prominent partial condition in cows affected with parturient paresis.

Symptoms.—Paralysis associated with lesions in the esophagus leads to the accumulation of dry food in the form of a cylindrical mass reaching from the stenosis to the pharynx (see choke). Unless this can be avoided, death from inhalation pneumonia occurs in a week to ten days. When the pharynx is paralyzed, one cannot easily determine that the esophagus also is involved. If ingesta passes the pharynx and accumulates in the esophagus the former is clearly excluded. It is probable that central paralysis affects both, and that peripheral paralysis (post-strangles paralysis) is circumscribed.

Treatment.—Whenever there is evidence of paralysis of the esopha-

gus from any cause the patient should not receive solid food, since it tends to pass down the trachea and cause fatal pneumonia. The symptomatic treatment is the same as for paralysis of the pharynx.

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VOMITING

Vomiting is the forcible expulsion of the contents of the stomach through the mouth or nose. It is under the control of a special center located in the medulla. It may be induced by causes that act upon the vomiting center, such as disease of the brain, and drugs like apomorphine—central vomiting; or by irritation of the afferent nerves of the stomach—reflex vomiting.

In *equines* the act of vomiting indicates a serious gastric dilatation. It may be due to primary indigestion from overloading, or secondary, due to obstruction at the pylorus (bots) or in the small intestine (torsion). The symptoms are those of a severe colic associated with retching, distress, sometimes squealing, and the discharge of sour-smelling contents from the nose. It should not be confused with choke in which food is also expelled through the nostrils with retching. *Treatment* consists in passing a stomach tube to relieve the gastric dilatation (see indigestion).

In *bovines* vomiting is less serious. Here, also, it is usually reflex, and induced by fermentable foods—green clover, mouldy silage. Infrequently, vomiting may be caused by traumatic gastritis. It may be central from disease of the central nervous system, from plant poisoning (*Veratrum viride* in the hay), or from drugs (veratrine, apomorphine). According to the cause, the act of vomiting may be free from marked distress, as after eating soft fermentable food, or it may be violent and painful. The vomitus is expelled in large quantities through the mouth. Marsh[1] writes of "spewing disease" in cattle and sheep in Utah; it is caused by sneezeweed and characterized by severe vomiting. He also mentions vomiting in sheep as one of the symptoms of black laurel poisoning. Persistent vomiting has been observed in 2- to 3-months-old calves affected with gastrointestinal catarrh and diarrhea. Stewart[2] reports that in more than thirty years' practice he has encountered only six cases of vomiting in cattle; in each case he found a foreign object or the trace of one in the esophagus.

Swine vomit easily from slight irritation in the pharynx or stomach; the vomiting center is relatively sensitive. It is a common symptom of indigestion, hog cholera, and swine erysipelas.

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ACUTE INDIGESTION IN RUMINANTS

(Overloading of the Forestomachs; Impaction of the Rumen; Impaction of the Omasum; Impaction of the Abomasum; Acute Tympany; Bloat; Atony of the Forestomachs; Dilatation of the Rumen)

Definition.—Acute and subacute indigestion of ruminants is an atony of the forestomachs induced by overloading of the rumen and reticulum with food or indigestible substances; depending on the kind of material, it leads to impaction of the rumen, tympany, or gastro-intestinal catarrh. In rare cases an excessive amount accumulates in the omasum or abomasum.

Etiology.—(*a*) *Overfeeding* is one of the most frequent causes. The value of a dairy cow is in direct proportion to the amount of food she is able to assimilate, and the digestive system is often crowded beyond capacity. Among the foods and combinations responsible, are clovers and timothy, corn fodder, grain, silage and hay, roughage and concentrates in any form. This type of overfeeding is common in cows on test for advanced registry; such cows are usually under careful supervision, however, and unless the food is decidedly abnormal the condition is recognized before grave symptoms develop. Cows under heavy feeding for production have an increased susceptibility to indigestion from eating damaged roughage or other imperfect food, and they are more severely attacked. Damaged alfalfa hay that can be consumed by the average cow with no ill-effect, may cause severe indigestion in a cow on advanced registry test.

Often overfeeding is due to ignorance and carelessness; animals are given fodder that is indigestible because of an over-ripe or heated condition, or are fed at irregular intervals—a common fault among inexperienced farmers. Many cases that occur in the first quarter of the year without apparent cause, belong in this group; the cows are overfed on dry fibrous roughage.

(*b*) Unlimited eating of food to which they are not accustomed is perhaps the most frequent cause of fatal indigestion in ruminants. They break loose from the pasture or stable and eat without restriction cornmeal, apples, green or partly cured corn fodder, recently cut heated silage, alfalfa, Ladino clover, green grain (oats, barley, buckwheat), ground feed (oats and barley) and newly threshed grain (wheat, buckwheat).

(*c*) *Indigestible and damaged food* is a less common cause: hay

mixed with chaff, frozen grass or silage, coarse dry corn-stalks, bean pods, straw contaminated with manure, bedding, rotten or moldy silage, old straw from the stack, buckwheat straw and sorghum.

(*d*) *Indigestible Substances.*—A cow often eats her own placenta, which may result in a severe attack with acute bowel catarrh or enteritis. Balls of hair and wool and a variety of metallic substances rarely may cause chronic trouble. Roughage, when heavily contaminated with sand, mud, or dust, is especially harmful.

(*e*) *Change of food* often leads to a disturbance of the digestive functions. The following examples are not infrequent: clover to swamp hay, marked increase in the grain allowance, from short pasture to full stable rations at calving time, and from dry stable fodder to luxuriant grass in the spring.

(*f*) In very *young animals* acute affections of the forestomachs are frequent. In calves up to six months of age, indigestion is often caused by eating substances that cannot be digested in the undeveloped stomach—silage, cornfodder, alfalfa, straw and shavings.

(*g*) *Predisposing causes* are acute bowel catarrh, ravenous appetite, advanced pregnancy, general weakness, adhesions between the reticulum and abdominal wall (healed traumatic gastritis), fatigue from long drives, and unaccustomed exposure to cold. Cows that are cast and that hang in the slings develop tympany, and if they remain cast for a few hours death will result.

(*h*) *Seasonal Influences.*—Nearly all of the cases that have been treated in the ambulatory clinic at Cornell University were in the months from October to April, its greatest frequency being in October and November. This seasonal occurrence is due to a change in the food. Cattle go from a dry pasture to a wide variety of green and partially cured roughage; they break into other fields, or eat roughage not normally relished.

Increased tendency to bloat on alfalfa from exhausted soils has been reported by McIntosh,[1] who believes it depends on chemical factors within the plant itself; he also mentions heavy losses in Ontario following prolonged dry spells, and the danger from wilted forage on excessively warm days. Increased danger from wilted alfalfa has been reported by Quin,[2] who observed an increased rate of bloat in sheep pastured on alfalfa in the afternoon; this was found to be due to an increase in the sugar content of the forage from 2.5 per cent in the early morning to 6 per cent in the late afternoon, when fatal bloat was definitely more frequent.

Morbid Anatomy.—In calves that have died of indigestion the foreign material is found in the rumen; its walls are congested and

slightly edematous. In overloading with heavy ground feed in cattle the rumen and reticulum show large areas black with congestion and hemorrhage; their walls are thickened and friable; the mucosa of the omasum is hemorrhagic, while the abomasum and small intestines are either congested or severely inflamed. The serous membranes of the heart may show hemorrhages and the heart muscle may be degenerated. Fatal overeating of heated corn silage or spoiled food leads to gastroenteritis. The autopsy shows a serous exudate in the abdominal cavity, degeneration of the parenchymatous organs, and congestion and hemorrhage of the abomasum and small intestines. In overloading of the abomasum with hay and grain there is an enormous distention of the fourth stomach, with gastroenteritis and secondary septicemic or toxic lesions. In death from chronic atony of the rumen the autopsy lesions are slight. Fatal bloat leaves extreme distension of the abdomen with the diaphragm extending far forward into the thoracic cavity; death is due to asphyxia.

Symptoms.—There is a history of anorexia and dullness, scanty evacuations, suspended rumination, sometimes vomiting, and tympany according to the character of the food. The milk flow is diminished. Animals not stabled usually have broken into another field and gained access to corn fodder, apples, etc., or in the late fall have eaten frozen grass. In the usual case, a systematic examination reveals dullness, normal mucosa, pulse from 60 to 100, the higher rate in those that have eaten fermentable food (corn fodder, apples, clover, frozen grass, filthy bedding), or an excessive amount of heavy food, such as oats and barley. About 20 per cent carry a pulse rate of 90 or higher. The respirations vary from 20 to 30, and they may reach 60 or more; the higher rate is most apt to occur when the contents of the rumen ferment. The temperature, except in mild cases, is 102°-104° F.; in the unthrifty it is often below 102°; corn fodder, clover, or cabbage carry it to 103° and above. After eating apples it is frequently subnormal (95°-98.5°); while in animals that are very sick from eating ground feed, it may be normal. In these very severe cases evidence of the grave condition is always present, however, in the form of cold extremities (horns, ears, teats), paralysis of the rumen, and a fast pulse. Chills are not infrequent, especially in the beginning. In severe forms pain may be manifested by an expiratory grunt or moan; it may cause restlessness, sweating, stamping with the hind feet, switching the tail, or kicking the belly; about 10 per cent show pain, either by grunting or restlessness. Other occasional general symptoms are stiffness, dry muzzle with mucous nasal discharge in severe types, weakness of the hind parts, swelling of the eyelids, lachrymation, and grinding of the teeth. A staggering gait is

characteristic of indigestion from apples. Coma-like symptoms are not infrequent; the animal is found in the attitude of milk fever, is unable to rise and lies with the head resting on the chest; this type has been observed in indigestion on corn fodder, apples, and ground feed. In calves opisthotonus and convulsions are occasional and the following nervous derangements have been observed in cows: paralysis of the pharynx, staggering and falling backwards, unequal dilatation of the pupils, and complete paralysis of the hind parts (paraplegia). Cases of this nature that terminate fatally may reveal inflammation of the stomach or intestines with a secondary encephalitis or distension of the brain ventricles with fluid.

The abdomen is found to be increased in size in overloading with roughage and in tympany. After eating green foods to which the animal is not accustomed (alfalfa, buckwheat, green corn fodder, apples), tympany often becomes the leading and dominant sympton. The abdomen is greatly enlarged, its wall tense and tympanitic in the left flank; the skin is moist with sweat; dyspnea becomes severe, the nose is extended, mouth open, tongue protruded and salivation abundant; restlessness or delirium and great anxiety are always present.

According to Cole[3] and associates the tympany following the eating of green foods, such as alfalfa and Ladino clover, is not due to excessive gas formation, but to a lack of scratching or irritation of the mucosa of the rumen by fibrous roughage. Such action has been shown by Schalk and Amadon[4] to induce regurgitation, and they were able to initiate it artificially by rubbing a wisp of hay over the inner walls of the rumen. Preventive measures consist in the introduction of sufficient fiber in the ration to initiate belching. In 1943 Quin[2] demonstrated that sheep with paralysis of the rumen were still able to eructate gas, and "that bloating in ruminants was frequently associated with a frothy mass in the rumen attributed to the rapid fermentation of sugar in the presence of saponin in the alfalfa on which the experimental animals were fed. This foamy consistency of the ruminal mass was held to be responsible for the difficulty in the eructation of gas"; even when ingesta was kept standing at room temperature the gas bubbles collapsed only after several hours. Dangerous production of gas could be controlled by feeding other materials first; it was believed that this prefeeding worked in a mechanical way on the texture of the ruminal contents by preventing the formation of a frothy glutinous mass and allowing the gas to escape. These observations are not in accord with the theory that bloat is prevented by stimulating ruminal activity.

When the contents of the rumen are dry (ground feed), the abdomen is normal in size and after two or three days it may even be smaller than

normal. On palpation of the rumen in the left flank the consistency is found to be increased; it may be doughy, though often it is firm. The contractions are weak and slow, from one per minute to entire absence in severe forms. Fermentation sounds are usually present, and complete absence of sound, especially to the phonendoscope, is a grave symptom. On percussion, or kneading with the fist, it may be possible to recognize pain over the rumen in the left subchondral region or over the reticulum in the region of the elbow. Other organs where pain may occasionally be induced are the omasum over the lower third of the right side between the sixth and ninth ribs, and the reticulum or abomasum behind the ensiform cartilage on the ventral surface; rarely one observes pain over the liver or spleen (referred pain). Pain induced by percussion over the omasum or abomasum suggests an overloading of these organs. And when a rectal examination fails to reveal tympany or impaction of the rumen, one may conclude, through exclusion, that the omasum or abomasum are possible seats of impaction and congestion.

In the right abdomen (small intestines, cecum, colon) the peristalsis is suppressed, but it may be increased in acute bowel catarrh from eating spoiled food, straw mixed with manure, filthy bedding, etc.

The bowel evacuations are scanty, and the feces are dark and firm. In the young, and in the presence of bowel catarrh or enteritis the feces may be fetid, mixed with mucus, and soft. When indigestion is complicated with bowel catarrh, the feces may be fetid, covered with mucus, filled with gas, and thin. Rectal examination reveals the size and consistency of the rumen; frequently it is distended towards the right side and into the pelvis by an excessive bulk of food. One also obtains evidence on the amount and character of the contents of the small intestines, cecum, and colon. Impactions of the small intestines are infrequent, but are easily recognized by palpation through the rectum. When making the rectal examination, one should also note the condition of the kidneys, the ureters, and the bladder, for disease of these organs has been diagnosed as "indigestion."

Course and Termination.—Usually recovery occurs in from one to three days. A more prolonged course may be due to neglect, the severity of the attack, or failure to withhold food. With the development of improved legume pastures reports of death from bloat have been increasingly frequent. Unrelieved acute tympany may terminate fatally in from one to three hours. Calves may die in convulsions within twelve to twenty-four hours after the first symptoms are observed. Overloading with heavy ground feed may terminate fatally in from three to four days, and heated corn silage or spoiled food leads to gastroenteritis. With prompt treatment the mortality from indigestion is low except

among sheep and calves. Coma-like symptoms early in the attack may lead one to anticipate a bad prognosis; but in the absence of other grave signs, and with active stimulation, the sensorium may become normal within a few hours. If no improvement occurs within three or four days after extreme overloading, the prognosis is unfavorable; when neglected the rumen and reticulum become paralyzed and gastroenteritis develops. Impaction of the rumen with a firm mass of food may lead to a chronic or incurable atony of the rumen; this is not frequent. Cows affected with chronic atony appear to improve, but upon eating the symptoms of indigestion recur because of weakness of the rumen; death from exhaustion is the final result.

Impaction of the abomasum is not easily diagnosed, and it is rarely found on autopsy. It occurs after eating dry soft material such as dried rotten straw, coarse bran, or chaff. It may be associated with symptoms of paresis and complete anorexia over a period of a week to ten days, finally ending in death.

In calves up to six months of age indigestion is serious because coarse roughage or foreign substances in the rumen are not readily expelled. Sheep are equally sensitive to the effects of overloading. This is probably due to the thinness and relatively weak contractile force of the muscular coats of the rumen.

Diagnosis.—Usually acute indigestion can be diagnosed by the history of faulty eating followed by anorexia, suspended rumination, atony of the rumen, and scanty evacuations. At first it may not be readily differentiated from traumatic gastritis; this is especially true of the severe forms with no obtainable history of faulty eating, and that show pain on percussion or kneading. Traumatic gastritis responds poorly to treatment; improvement is incomplete or is soon followed by another attack; the loss in condition is more marked, and may progress when the acute symptoms of indigestion recede; in advanced pregnancy parturition is apt to aggravate traumatic gastritis, whereas, in simple indigestion parturition is followed by improvement. Traumatic gastritis with acute diffuse peritonitis may be diagnosed as primary indigestion. Diffuse peritonitis rarely develops from a primary indigestion. The symptoms that indicate peritonitis are: complete and persistent anorexia, scanty watery or mucous evacuations after administration of laxatives, failure to improve after the rumen and intestines have been stimulated to contraction, tympany several days after the onset due to accumulation of gas between the rumen and the abdominal wall (tympany of primary indigestion occurs at the onset, later it is not present), pain on kneading over any part of the abdomen, shallow breathing, painful expiratory grunt, recumbency, stiff careful movements when

walking, slight daily increase in the pulse, and sudden decrease in the milk flow. Animals kept on natural pasture rarely suffer from primary indigestion, so that a severe indigestion in such individuals is suggestive of traumatic peritonitis.

Secondary atony of the forestomachs is symptomatic of nearly all acute disturbances in cows; it may be merely a symptom of fever; it is nearly or quite complete in peritonitis, in intussusception, in severe metritis or mastitis, in the digestive type of acetonemia; and it is a constant symptom of acute affections of the respiratory and digestive systems. In stabled cows, with no history of change in diet, primary indigestion with colic may resemble the onset of either intussusception or pyelonephritis.

Treatment.—In all cases of ordinary simple indigestion the chief object is to reestablish contractions of the rumen and to evacuate the digestive tract. To avoid more serious trouble all food should be withheld until both of these conditions have been met, for a relapse is not infrequent when the patient is fed. The average case responds promptly to a laxative combined with antiferments, carminatives, and stimulants. The chief causes of death in primary indigestion in bovines are gastroenteritis, caused by extreme overloading, or suffocation from acute tympany, caused by extreme overloading and fermentation.

When called to treat a *bloated cow* the owner should be advised to put a gag in the mouth of the animal, and stand the cow with rear parts lower, or force the patient to move slowly up and down hill until assistance arrives. On arrival the first object is to determine whether rumenotomy is required to evacuate the contents and relieve symptoms of impending death from suffocation. While the tension may usually be relieved through a stomach tube or trocar, this treatment may fail if the gas is intimately mixed with the food in the rumen (frothy bloat). In deciding whether or not rumenotomy is indicated one needs to consider that removal of the contents through the flank is a safe operation, and that failure to operate may result in death.

After passing the stomach tube it is common practice to introduce warm water, 5-10 quarts, to which has been added one or more of a variety of laxatives, antiferments, and carminatives. Thus Fincher[5] administers tarter emetic 2.5 drams (12 Gm.) dissolved in several liters of warm water and pumped into the rumen through a colt stomach tube passed through the nostril, or a ¾-inch stomach tube passed through an Emont stomach-tube speculum. To this may be added one or more of the following: aromatic spirits of ammonia 1 to 3 ounces (30-90 cc.); fluid extract or tincture of capsicum 1 to 4 drams (4-16 cc.); turpentine 1 ounce (30 cc.); soluble pine oil 1 to 2 ounces, or creolin 1 to 4 drams

(4-16 cc.). One may also give mineral oil (4000 cc.), which is said to have an added advantage of increasing the surface tension of the gas bubbles. Other preparations currently recommended are milk of magnesia ½-1 gallon (Emblo powder 1 lb.) or sodium hyposulfite ½ to 1 ounce (15-30 Gm.). In 1949 Quin and Austin[6] reported the value of methyl silicone (Tympanol) in the treatment of tympany. This is injected directly into the rumen through a needle or diluted with water and given orally. As postulated by Quin and Austin, bloating is primarily due to gas entrapment within froth bubbles because of decreased surface tension. It is said to increase the surface tension of each tiny gas bubble contributing to frothy bloat, and permits them to increase in size until they rise to the surface and may be eructed in a normal manner. In chronic tympany, relief may follow the use of a large quantity of rumen contents of another freshly slaughtered animal mixed with water and pumped into the rumen through a stomach tube. Stimulation of the contractions of the forestomachs is also aided by the administration of strychnine sulfate (1-1½ grains) orally three times a day, but this should not be administered to cattle subcutaneously.

Contractions of the rumen may be stimulated by means of massage alone; with closed fists knead the left flank thoroughly from below upward for from fifteen minutes to one-half hour. In obstinate cases, when the actual condition cannot positively be determined, and the continued use of laxatives seems indicated, it is well to select one that is effective and non-irritating, such as mineral oil, for the strength of the patient must be carefully guarded.

The feeding of dry roughage before turning cattle into luxuriant pastures tends to prevent bloat; the mode of action is explained by Clark[7] who observed that the addition of chopped hay to foaming ingesta breaks down the froth. A forecast of the introduction of methyl silicone in the treatment of frothy bloat is found in a statement by Clark[7] that "turpentine is still the best drug for the treatment of acute bloat but as it acts largely through its effect on surface tension superior preparations may yet be found." Also "It is very doubtful whether disinfectants should be introduced into the rumen even if they are effective in reducing gas formation. A properly balanced and active microflora is essential to ruminal digestion and its impairment or destruction may have serious sequence." Observations from practice, however, do not confirm the theory that disinfectants as temporarily used in the treatment of acute tympany impair the digestive functions of the ruminal bacteria.

Excessive impactions, found in cows that have accidentally had access to corn fodder, grain, or meal, and have overeaten so heavily that

contractions and evacuations cannot be accomplished by the usual method, require prompt and effective measures. If the contents are dry and heavy with little fermentation they may be softened by pumping several pailsful of warm water containing carminatives into the rumen through a stomach tube. Then follow with massage of the left flank by means of kneading movements with the closed fists carried from below upwards. In a few minutes eructations occur, contractions of the rumen are reestablished, and gradual improvement follows. One should also administer laxatives, carminatives, and stimulants as in the usual type. This method may be effective, or, in overloading with a heavy fermentable mass such as alfalfa or green corn, it may be too slow; one must then resort to rumenotomy with removal of the contents through the flank. This is described in the chapter on traumatic gastritis. Frick[8] has reported its use on 15 animals, with 14 recoveries, in a group of steers that over-ate on cracked corn and cottonseed cake.

In impending death from tympany and suffocation, one may only have time to gash through the flank with a knife. The contents of the rumen rush out with considerable force, and while the life of the patient may be saved, healing is prolonged and usually leaves an adhesion between the rumen and the abdominal wall; but an adhesion here does not interfere seriously with digestion.

Paresis may respond as promptly as milk fever to inflation of the udder with air. In this type one also gives laxatives and stimulants, using a stomach tube for oral medication.

In coma, weakness and poisoning following unlimited consumption of half-ripe corn on the cob and green cornfodder, Fincher[5] has reported favorable results from sodium hyposulfite 30 per cent and sodium nitrite 2 per cent (50 cc.) per vein, when the usual treatment consisting of large doses of calcium gluconate intravenously had failed. The coma of indigestion from apples or green corn is usually overcome by calcium gluconate (1000 to 1500 cc. of a 40 per cent solution) per vein, and this may be supplemented with dextrose (500 cc. of a 20 per cent solution). In any severe form of indigestion the administration of large doses of dextrose and calcium gluconate may be highly beneficial.

Following recovery from the acute symptoms of indigestion, a cow may have anorexia or appear dull. This condition usually responds to bitters, such as:

℞ Sal Carolini factitii	℥ xvi (500 Gm.)
Gentianae	
Nucis vomicae	aa ℥ viii (250 Gm.)

M. Sig—One-half ounce (30 Gm.) thrice daily.

Or one may prescribe a grain of strychnine sulfate to be given orally three times a day. The following mixture of equal parts 1 per cent solution of arsenic trioxide and 1 per cent solution of strychnine sulfate 4 drams (16 cc.) thrice daily is excellent. The two solutions are made as follows:

Arsenic trioxide	5 Gm.
Acid hydrochloric, dil	3 cc.
Water to make	500 cc. or
Strychnine sulfate	10 Gm.
Acid hydrochloric dil	20 cc.
Water	980 cc.
Heat	

Mix these solutions in equal parts and give one-half ounce (15 cc.) two to three times daily.

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TRAUMATIC GASTRITIS IN RUMINANTS

(Traumatic Peritonitis; Pericarditis; Hepatitis; Splenitis)

Definition.—Traumatic gastritis includes a variety of lesions that follow perforation of the reticulum, less often the rumen, by a sharp object. In every case there is a peritonitis; often there is a pericarditis and pleuritis; while less frequently the liver, spleen, or lungs are involved. It is of chief importance to distinguish between peritonitis and pericarditis; the latter is more easily recognized and is highly fatal, while peritonitis is more obscure and may yield to treatment. This disease causes a heavy loss in many herds. In the ambulatory clinic of the New York State Veterinary College it is one of the chief causes of death in cows. Because of its similarity to indigestion and other diseases it is under almost constant consideration.

Etiology.—Perforation of the reticulum is favored by its small size, the honeycomb-like structure of its mucous surface, the activity of its contractions, and the tendency of cows to swallow foreign objects that remain in the lower anterior part adjacent to the diaphragm and pericardial sac. It is here that injury is most frequent. Most adult cows have pieces of metal in the reticulum, and many slaughtered old cows reveal injuries from such foreign bodies. These injuries may result in local inflammation and healing without causing serious symptoms; the cow recovers from what is perhaps diagnosed as a severe indigestion. Such cases terminate in a *chronic adhesive circumscribed peritonitis* between the reticulum and diaphragm. This may cause no further trouble; but increased pressure from advanced pregnancy or parturition may advance the body and extend the fistulous tract, thus inducing serious and extensive lesions in adjacent organs. *Advanced pregnancy* is a part of the history of at least 50 per cent. In some cases, the observed symptoms date from parturition; but more often the animal has not been normal during advanced pregnancy, and the symptoms increase after delivery. In a four year period the monthly distribution of cases diagnosed in our ambulatory clinic as traumatic gastritis was as follows:

January	11	July	8
February	8	August	4
March	22	September	12
April	12	October	9
May	18	November	8
June	14	December	14

The high rate in March, April, and May is probably explained by a corresponding parturition rate. In relation to *age*, it is seen chiefly after two years, but it is not rare in yearlings. Males are often affected, especially old bulls that have been exposed to careless disposal of baling wire. Traumatic gastritis is most prevalent on farms where baled hay is used, wire fences are allowed to fall and disintegrate, rubbish is thrown into the pasture and lanes, and repairs are being conducted in the stable.

Morbid Anatomy.—The anatomical changes and the foreign body are most readily found when the cadaver lies on the right side and is opened on the left side. Carefully remove the digestive organs with the exception of those around the anterior part of the forestomachs. Then explore the interior of the reticulum for foreign bodies, and look for adhesions between it and the diaphragm. Changes that have spread from this area are readily traced to the neighboring organs in the abdomen and thorax. Abscesses are found in the body cavities near the

diaphragm, in the spleen, the liver, and the lungs. When the heart is involved the pericardial sac usually contains thin fetid dark-colored pus, or pus and fibrin; sometimes the heart muscle itself is injured. The sharp object may have disappeared through erosion, but the adhesion between the reticulum and diaphragm remains as proof of the injury.

Symptoms.—Four chief types may be recognized: (1) Acute circumscribed peritonitis, (2) Acute diffuse peritonitis, (3) Chronic peritonitis, and (4) Pericarditis.

Acute Circumscribed Peritonitis.—This is the most common form and it is the initial lesion in nearly every case. When the object is long and sharp, a diffuse peritonitis or pericarditis or both may immediately develop, but as a rule the lesion is at first circumscribed. From the initial injury it may extend in a widely variable manner. The onset is sudden and marked by loss of appetite and milk flow; a sudden and almost complete cessation of the milk secretion is a distinctive symptom. Vomiting has been observed at the onset of traumatic gastritis, but it is rare. Often the owner reports a diminishing appetite and milk flow over a period of two to four days; less often there is a history of repeated indigestion, or a peculiar stiffness. On examination, one usually finds depression, arched back, and stiffness. Habitual recumbency is frequent. Trembling of the muscles behind the elbow is common, and this symptom is regarded by some as almost pathognomonic of traumatic gastritis; it occurs, however, in many other conditions. Often the elbow is abducted. The eye is slightly sunken and has an anxious expression. The mucous membranes are normal. In the typical case the pulse is 75 to 100, the respirations 30 to 40, the breathing shallow or irregular, and the temperature 102° to 104° F. The higher rates suggest either diffuse peritonitis or pericarditis, especially when associated with a short course and severe pain. Directly after the perforation there develops an acute peritonitis, and with it a rise in temperature. As the area of peritonitis becomes walled off, the temperature recedes. The usual elevation is from 103° to 104° and a fraction. In abscess formation in the spleen, liver, or lungs, the temperature tends to be higher. In any case, it is variable throughout the course of the disease. It may remain normal or only slightly elevated in the less acute types. The abdomen is normal in size, the contractions of the rumen are weak and infrequent, and bowel evacuations are usually suppressed; there is rarely diarrhea. Pain is induced by percussion or kneading with the fist over the region of the reticulum or the xiphoid cartilage or by pinching the back in the dorsal region. Occasionally there is pain on percussion at some distance from the injury—referred pain. When forced to move, the animal usually does so reluctantly. If a suspected case walks

actively and jumps freely over a gutter or a curb when entering a box stall, probably it is not traumatic gastritis. In some instances there is no pain on percussion; this may be explained by the deep location of the injury. Infrequently, a circumscribed painful swelling appears at the left of the xiphoid cartilage; this indicates an abscess, a previous course of several days or weeks, and a peritonitis that is probably circumscribed.

The *course* depends on the degree of injury to the peritoneum. Small sharp objects, such as 6-penny nails, short pieces of wire, and pins cause severe symptoms at the time of the perforation. But they are walled off in 3 or 4 days by the inflammatory reaction that leads to permanent adhesion between the peritoneal surfaces. The acute symptoms may then promptly or gradually recede and result in a complete or partial recovery. Recurrent attacks are not infrequent; often these are caused by a short nail that injures repeatedly. The acute course is three or four days to a week.

After an acute attack, infection, sometimes the object itself, may pass to the *liver* or *spleen*, causing abscess formation. In splenic abscess there is a tendency to metastasis to the joints and tendon sheaths, the lungs, and the liver. In these cases the animal usually dies or is slaughtered after a few days to a few weeks.

Acute Diffuse Peritonitis follows perforation with a long wire or nail; the inflammation is not walled off and soon it becomes extensive. The onset is sudden and the general symptoms are marked—pulse 80 to 100, breathing 50 to 60, and temperature 104° to 105°. On percussion, or kneading, abdominal pain is severe. Treading movements with the hind feet, grunting and other signs of acute pain may be present. Death occurs in from ten to fourteen days. In explanation of a course of ten to fourteen days in acute diffuse traumatic peritonitis, Dr. Gibbons has expressed the view that during the first few days a protective exudate forms in the region of the perforation, and that when this finally breaks down the course is only one or two days.

Chronic Peritonitis.—This varies widely from a circumscribed scar or abscess which causes no symptoms, to extensive adhesions of the peritoneum which cause emaciation; thus it may be circumscribed or diffuse. It may develop from acute inflammation or be chronic from the beginning. There may be a history of unthriftiness dating from advanced pregnancy, from an attack of "indigestion," or from a previous traumatic gastritis. Poor condition, stiffness, debility, and recurrent attacks are about the only general symptoms. In one case there was chronic bloat due to extensive adhesions of the rumen. A second suffered from an acute attack of traumatic gastritis and at parturition one year

later suffered from apparent metritis. Douching caused distress and dyspnea, and an autopsy revealed adhesions involving nearly all of the abdominal and thoracic organs. It is this chronic form that often flares up in association with other diseases, as metritis, mastitis, and indigestion, thus bringing about a confusing complication.

Pericarditis.—Fully half of the well-defined perforations of the reticulum extend through the diaphragm to the adjacent pericardium. The usual condition is a fistula uniting the two cavities. This is surrounded by a connective tissue cord, often by abscesses, and usually it contains the foreign body. The general symptoms are severe. Depression and loss of condition are marked. The mucous membranes are pale, and recumbency is the rule. The temperature may be normal, but usually it is raised—102° to 104° F. Compared with other forms, the temperature is higher in pericarditis; it may even go to 105° or 106°. To obtain an accurate record of the temperature in traumatic gastritis, it must be taken daily for several days; this reveals the frequent daily variation. There may be a terminal subnormal temperature. Characteristic symptoms are found on examination of the circulatory system. The heart rate is between 70 and 120; in over 50 per cent of the cases it is 100 or more. Pain on percussion can nearly always be demonstrated when the animal is able to stand and have the front leg carried forward so that percussion can be applied directly over the heart. Pain is more marked in the acute form. When the lesions have developed gradually, the pericardium has become thickened, and the sac filled with fibrin, the pain on percussion is less severe. Percussion may reveal dullness. The heart sound is usually modified. It may be splashing (liquid), tinkling (fibrin and liquid), diminished or absent (fibrin), or increased in direct injury to the heart. Occasionally the sound on auscultation is most marked on the right side. Distension of the jugular vein and edematous swellings in the region of the throat, neck or sternum are seen in this disease only in pericarditis and as a rule during the terminal week. The most useful diagnostic signs are modified heart sounds, rapid heart rate, pain on percussion, and an increased area of dullness. The condition is practically always fatal, and the course is from two to three days to a month or more. Extensive pericarditis may develop without attracting the attention of the owner.

Pleuritis may constitute the chief lesion. The course is from two to six months. There is a gradual loss in condition combined with digestive and respiratory symptoms which vary according to the location and extent of the changes—pleuritis, pulmonary abscess, bronchopneumonia. The temperature rarely goes above 103°. Respirations may increase. Emaciation becomes extreme and the animal is usually

slaughtered. The *respiratory symptoms are characteristic.* A large abscess in the lungs or pleural cavity gives dullness that is complete and sharply defined. Percussion over the chest is painful. Usually there is a chronic bronchopneumonia causing a variety of moist râles. Cough is constant; it is frequent, painful, suppressed and easily induced.

Pulmonary Emphysema infrequently occurs from perforation of the lung. Air escapes beneath the pleura, peritoneum, and under the skin, causing death from suffocation in from one to three days. The dominant symptom is subcutaneous emphysema extending over the entire body. Breathing is labored.

Diagnosis.—So many of the symptoms of traumatic gastritis are common to those of *primary indigestion* that an indefinite diagnosis is frequent, especially at the time of the first examination. In traumatic gastritis the symptoms are usually severe; in primary indigestion with severe symptoms the cause is usually obvious—accidental overeating or spoiled food. Primary indigestion usually responds to treatment in one or two days, while traumatic gastritis often continues for one or two weeks. In primary indigestion, tympany often occurs at the onset of the attack; in peritonitis, tympany occurs as a terminal symptom. There is a tendency to attach too great importance to the rectal temperature as a means of distinguishing between primary indigestion and traumatic gastritis. Because of infection, one might anticipate elevation of temperature as a constant symptom of peritonitis in cows. In infectious or pyogenic diseases in bovines, it is the rule that the rectal temperature varies widely and, judged by itself, a single reading is frequently an unreliable guide. Tabulations of temperatures in traumatic gastritis and primary indigestion show a more constant and higher elevation in the former disease. In primary indigestion the temperature is usually normal. Frequently one observes prompt recovery of cases in which the symptoms are like those of acute circumscribed peritonitis; it is probable that the majority of these are primary indigestion. For symptoms of chronic local pneumonia resembling traumatic gastritis, see p. 45.

Subacute or chronic pneumonia may be mistaken for traumatic gastritis on account of pain on percussion behind the elbow. A cow with pneumonia may even stand with the front feet crossed, a characteristic attitude in traumatic gastritis. The distinctive differential signs of pneumonia are rapid breathing and râles.

Peritonitis from other causes is most commonly met with in perimetritis, necrobacillosis, and tuberculosis. In fatal peritonitis following accidental puncture or rough manipulation of the uterus or rectum, the onset dates from the injury. An animal with a normal appetite suddenly develops pain, stiffness, depression, and complete anorexia. Fol-

lowing puncture of a uterus affected with acute metritis, death in less than a week is the rule; if involution of the uterus is complete, the uterus may be punctured without causing obvious reaction. *Peritonitis following parturition* sometimes closely resembles traumatic peritonitis or pericarditis. In general, any sickness at the time of parturition should be regarded as primarily an affection of the uterus until proved to be otherwise. Occasionally a chronic traumatic peritonitis flares up at this time; it may be suspected when involution of the uterus takes place, and yet the cow continues to be sick. In tuberculous peritonitis, a rectal examination will usually reveal marked enlargement and induration of the oviducts. Tuberculosis of the pleura and of the pericardium may cause physical symptoms practically identical with those caused by foreign bodies that penetrate the chest wall.

Necrobacillosis of the liver may extend to the peritoneum, causing symptoms similar to those of traumatic gastritis. It is more prevalent in two-year-olds; the temperature is higher and more constant, 104° to 106° F.; jaundice may occur if the liver lesions are diffuse, but usually they are circumscribed; there may be pain on percussion over the liver; and the lungs contain necrotic foci that may induce respiratory symptoms. Habitual recumbency is not observed. Death occurs within not more than two weeks after physical symptoms appear.

Multiple abscess of the liver has been diagnosed as traumatic gastritis. In one of these the cow showed progressive loss in condition and habitually stood with the front legs crossed during the preceding six months.

The *seasonal occurrence* often serves as a useful guide. While traumatic gastritis is not frequent in the summer months, we have rarely met with a case of primary indigestion in a cow on native pasture. Exceptions are observed when the cows are first turned into a luxuriant growth, and when coarse indigestible weeds and other roughage are grazed after a shortage of grass.

In the case of *thrombosis* around the auriculoventricular valves of the heart, seen chiefly in the right heart, the symptoms and course closely resemble those of traumatic pericarditis. The rate of the beat is high, but there are no abnormal sounds with the possible exception of a slight swish.

Chronic *painful conditions of the joints* have been mistaken for traumatic gastritis. The gradual loss in condition, the frequent shifting of position because of pain, and the tendency to remain down are the leading symptoms. Consider bilateral gonitis, and fractures, sprains, or injuries involving the pelvis, the pelvic ligaments, or the lumbar and sacral vertebrae.

Chronic bowel catarrh may closely resemble chronic circumscribed

traumatic peritonitis. The leading symptoms are poor appetite, gradual loss in condition, and soreness on pressure or pounding with the fist over the abdomen. If the animal is subject to recurrent diarrhea, it is probably not traumatic. In general, in affections involving the abomasum and duodenum, the appetite is more variable and the depression is less marked.

Pyelonephritis shows characteristic changes in the urine, and enlarged kidneys or ureters may be found on rectal and vaginal examination.

Ulcer of the abomasum resembles acute traumatic gastritis, and following rupture there is collapse with a pulse well above 100.

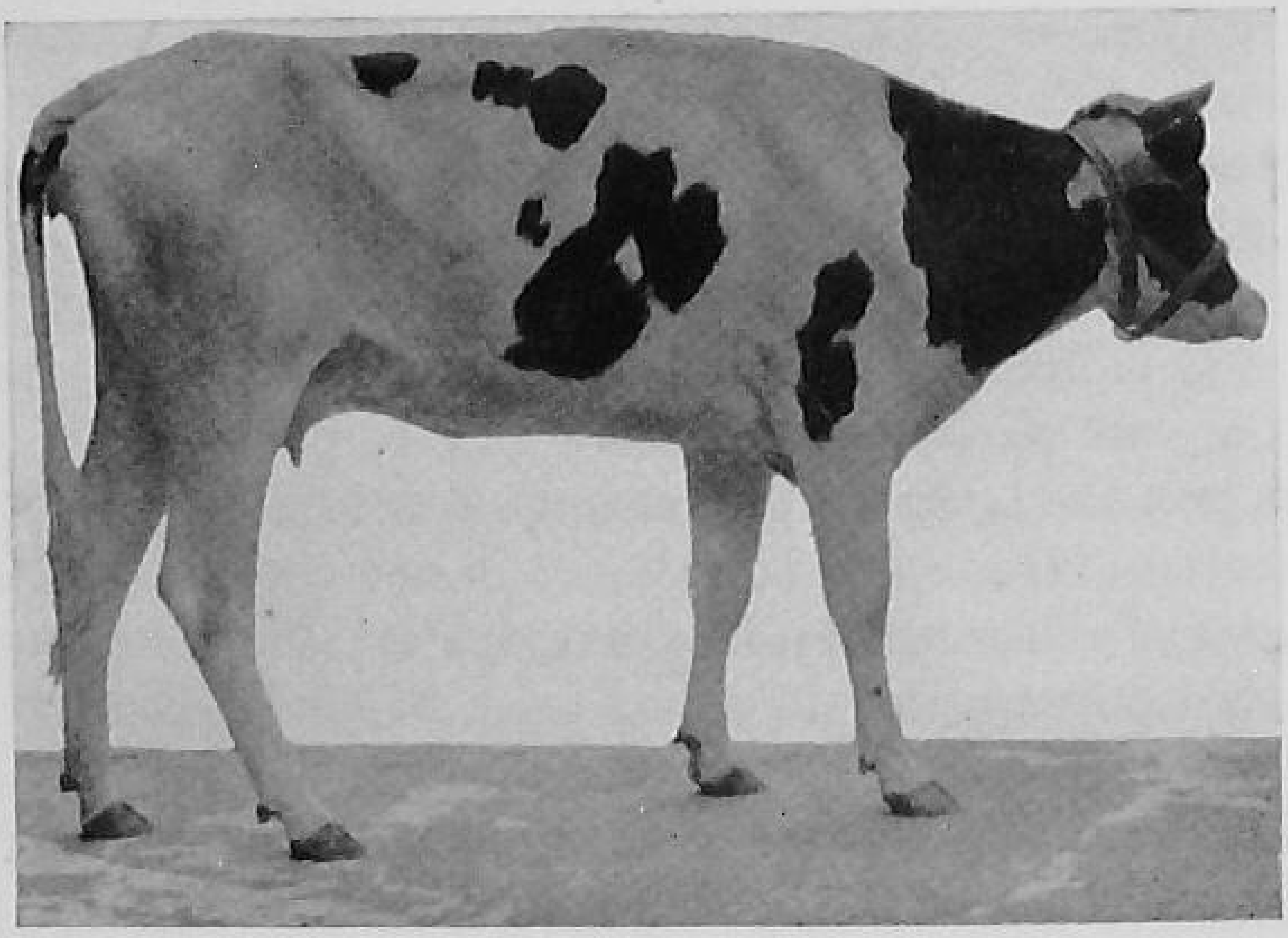

Fig. 10.—Advanced traumatic pericarditis. Note arched back, tucked-up abdomen, and painful attitude.

Acetonemia may present symptoms similar to those of traumatic gastritis in its occurrence shortly after parturition, loss of appetite and condition, and soreness on palpation. Differentiation may be made by the positive Ross test in acetonemia, but the Ross test may also be positive in traumatic gastritis.

An examination of the blood may show a marked increase in the neutrophiles, in association with any extensive purulent condition. In one case of traumatic gastritis, for example, the neutrophiles were 82 per cent in contrast with a normal of 10 to 30 per cent. When a blood smear is normal, one needs to hesitate before making a diagnosis of traumatic gastritis, on loss in condition, inappetance, and soreness on percussion.

Treatment.—As previously stated, pericarditis and acute diffuse peritonitis terminate fatally within a few days. As a rule, diffuse

chronic peritonitis is incurable, though it is not immediately fatal. This leaves *circumscribed peritonitis* as the only condition that may be relieved by means of treatment. When traumatic gastritis is suspected, one should avoid the use of laxatives that may irritate the digestive system and weaken the patient, such as magnesium sulfate, barium, tartar emetic, etc. Autopsy and abattoir reports show that circumscribed healed traumatic peritonitis is very common in dairy cows. Thus in borderline cases, where the distinction between primary indigestion and traumatic gastritis is not clear, and the general condition is not seriously disturbed, expectant treatment may be followed with safety. Relief may follow the use of a raised platform at an elevation of 8-10 inches under the front feet. This method has been widely recommended, but when recovery follows its use the diagnosis may sometimes be questioned. Because of the use of twine instead of wire for baling hay and straw, and the use of electric fences instead of permanent wire fences, there has been a decrease in the frequency of traumatic gastritis. But when the symptoms persist and the general condition and milk production remain poor, operative removal is indicated.

Operative removal of foreign bodies was recommended by Obich[1] in 1863. His method of operating on the standing animal was very similar to that in use today. Wenger[2] has reported that Obich's operation was performed by various practitioners and the results were not satisfactory. Failure was attributed to the selection of patients in which the disease was too far advanced. From their own experience, they conclude that early positive diagnosis is very difficult, and that the owners of animals are opposed to an operation. The effect of elevation of the front end of the cow by means of a slanting platform has been reported by Kolb;[3] this position is supposed to cause the sharp object to fall into the cavity of the reticulum. Another method has been described by Schöberl:[4] the cow is placed on its back and the abdomen just posterior to the sternum and on the left side, is kneaded with the foot. The method described by Schöberl has not proved to be generally effective. Elevation of the front end of the cow on a slanting platform is widely used, and it is claimed by Rüegg[5] to be the most important therapeutic measure. The front end of the platform should be not less than 8 inches high, and to prevent slipping narrow wood cleats or pieces of old rubber hose may be nailed crosswise over sacking; on a dirt floor the inclined surface may be built of this material. Reports on the efficiency of this method indicate that improvement may be only temporary.

Operative treatment for the relief of traumatic injuries has been reported in the United States by Bosshart,[6] and Bardwell.[7] Bosshart

reported 19 recoveries in 25 operated cases. Bardwell reported 9 recoveries from 12 operations performed during the period April 22 to July 14, 1927. Of the three that died, one was regarded as a case of incurable pericarditis before the operation was performed; the second proved to have diffuse peritonitis, as suggested by the symptoms (pulse 100, temperature 104.6°); the third was affected with degeneration of the liver, apparently of traumatic origin. Dr. Gibbons[8] has reported 29 recoveries in 43 operated cases in a period of five years, in the ambulatory clinic of the New York State Veterinary College. In a number of instances, we have done the operation when it was previously apparent that the disease had progressed beyond any

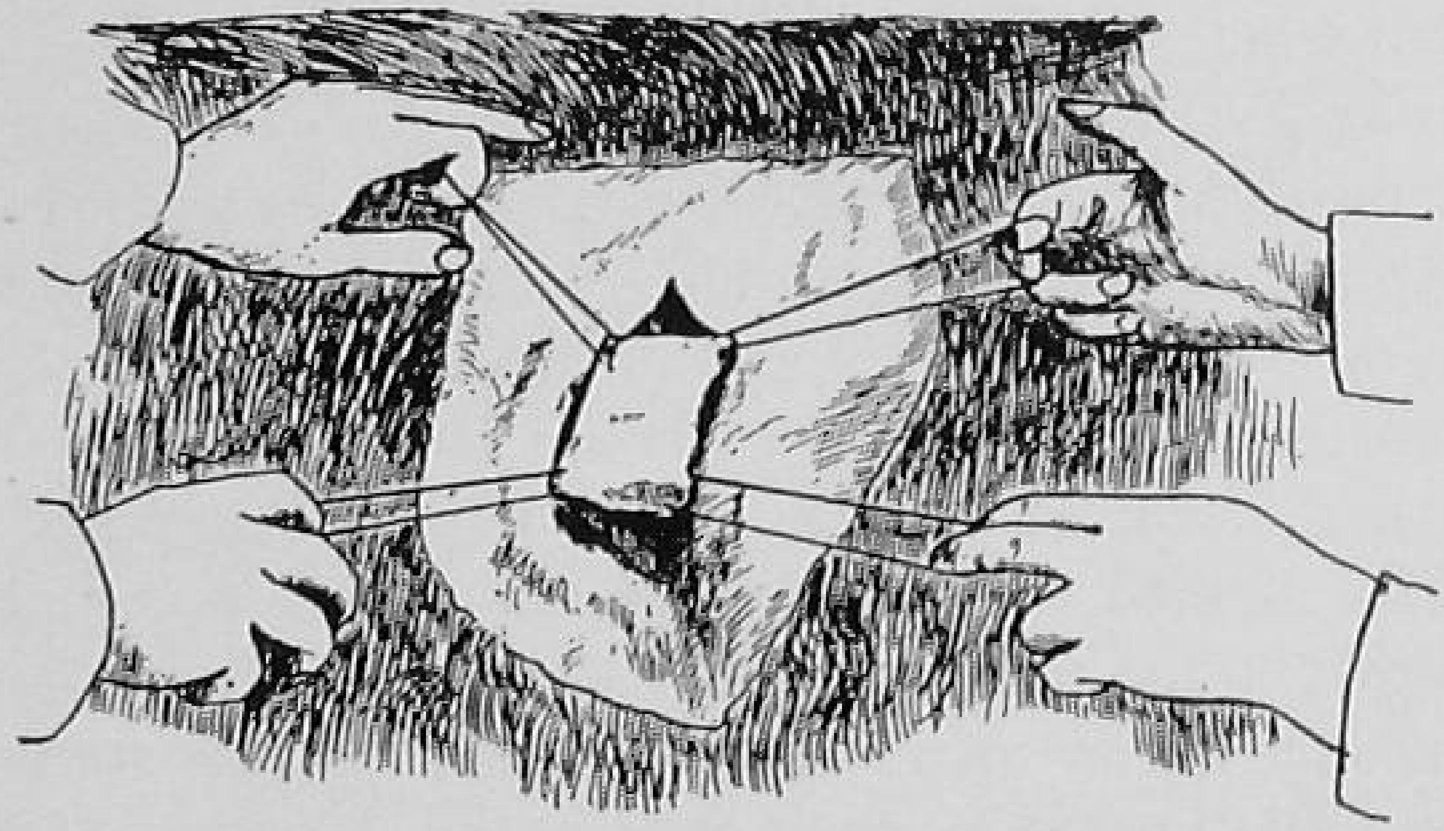

Figure 11

reasonable hope for recovery. From these reports it is evident that in circumscribed traumatic peritonitis accurate diagnosis and operative relief are frequently possible.

To summarize the symptoms of this condition, one considers the following points: (1) A sudden onset with symptoms of indigestion from no apparent cause—anorexia, suppressed rumination and peristalsis, and scanty bowel evacuations. (2) History of unthriftiness, stiffness, previous attacks, advanced pregnancy, or access to wire or nails—repairs, old fences, baled hay. (3) Pain shown by arched back, anxious expression, recumbency, restrained walking, by kneading behind the elbow or sternum, by pinching the dorsal vertebrae, or by the animal's grunting. (4) Trembling of the muscles in the region behind the left elbow. (5) Pulse of 80, respiration shallow, and temperature of 103° or higher and variable from day to day. (6) Medicinal treatment is ineffective, or leads to incomplete recovery. (7) Exclusion of other diseases.

If the diagnosis is positive, and the symptoms are not too suggestive

of diffuse peritonitis, pulse of 80 or more and temperature of 104° or higher, an immediate operation is indicated. If the temperature and pulse are high, it is better to administer mineral oil and stimulants for two or three days, or until the acute symptoms recede, before proceeding with an operation. If traumatic gastritis is only suspected, or highly probable, one may place the animal on an inclined platform and await developments.

This operation is now being performed with increasing frequency. It

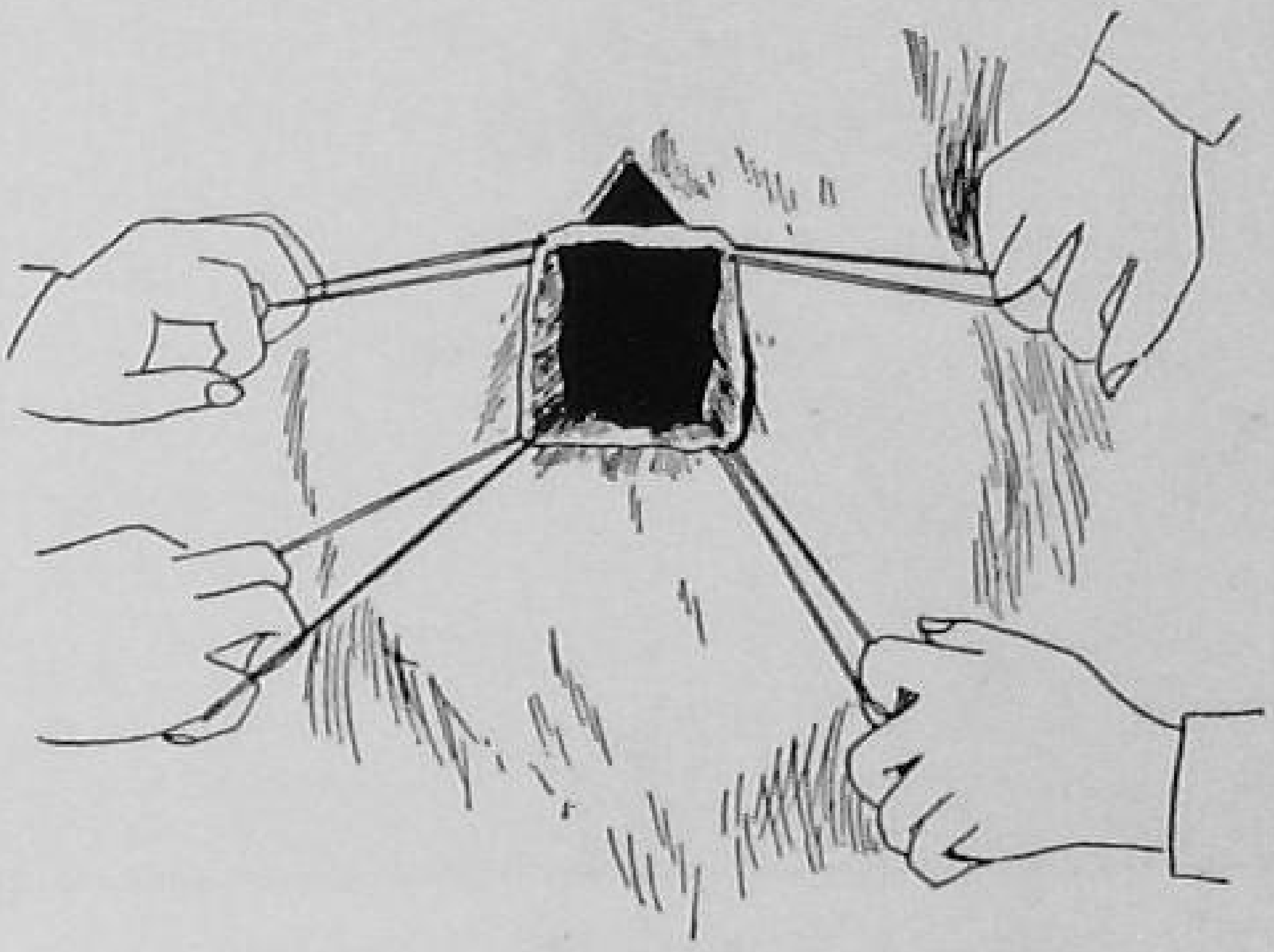

Figure 12

may reveal traumatic gastritis when there is no more evidence of its presence than a gradual loss in condition without apparent cause; and removal of a small wire or nail that is apparently harmless may be followed by immediate improvement.

The Operation.—This is done with the cow either in a stanchion or tied with a halter. The field of operation is shaved, and disinfected with a 1:500 alcoholic sublimate solution, or with ether, alcohol, and picric acid. About 75-100 cc. of a 1 per cent tutocain solution is injected under the skin and into the muscles. The opening in the skin is made about 6 inches long, and the muscles and peritoneum are cut to agree with the skin incision. A suture of umbilical tape is made through the wall of the rumen where it lies adjacent to the dorsal end of the incision in the abdominal wall. The ends are tied and the tape is held by an assistant. A similar tape is placed through the rumen, about 2 inches posterior to the first. Two more are then inserted at the ventral end of the incision. Two assistants, one at either side of the operator, now pull the rumen through the incision in the abdominal wall (Fig. 11). The rumen is opened and held with the sutures so that the contents do

not fall into the operative wound (Fig. 12). A rubber sheet attached to a metal embroidery hoop is used to protect the edges of the wound from

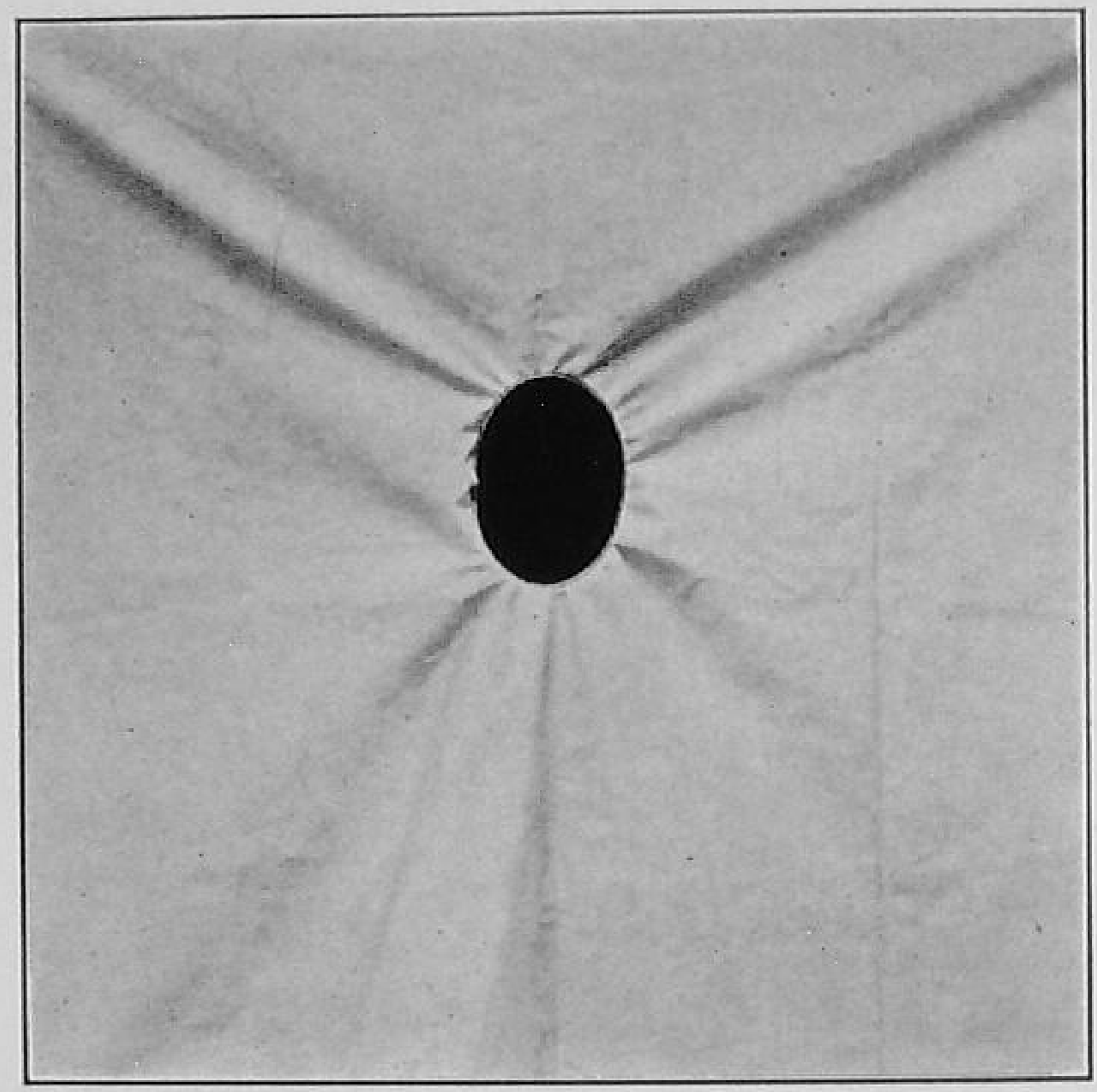

Figure 13

Figure 14

contamination while the contents of the rumen are being removed (Fig. 13). The ring is turned in a vertical direction and passed through

the incision into the rumen, where it is adjusted in such a manner that the contents may be removed through the rubber sleeve (Fig. 14). The compact material in the upper part of the rumen is removed. That in the lower part is semi-fluid, and is left, for it is thought to be of some value in maintaining the physiological contractions of the fore-stomachs. A search for the foreign body is then made. In the majority of cases the object which is causing the trouble is easily found. It may be necessary to make a careful search of the pockets in the mucosa of the reticulum with the end of the finger; it is located by feeling for the thickened adhesions that are usually around it. In removing the object, care should be taken not to tear down the adhesions. The rubber sheet is now removed. Some operators wear a rubber glove and sleeve until after the removal of the rubber sheet, and then remove the glove before the wounds are sutured; others prefer to operate without gloves until ready to apply the sutures. Some discard gloves entirely; in this case it is important to wash the arms thoroughly, and to disinfect them with alcoholic sublimate before suturing the wounds. The edges of the incision in the rumen and abdominal wall are wiped with dry cotton or gauze. The incision in the rumen is closed with two rows of Lembert sutures, No. 3 20-day chromic catgut, using a double throw square knot to prevent slipping. The top suture is left with long ends to be held by an assistant while the first row is inserted. The second row is made in a like manner. The peritoneum and muscles are then sutured together with catgut, placing the sutures close together. The peritoneum is not sutured separately. The skin is closed with mattress sutures of linen. The incision is covered with cotton or gauze. Usually the stitches are removed one week later. If infection develops, the incision may be opened to permit drainage. Keep the cow quiet and avoid bulky food for two or three days. Stitch abscesses frequently develop, but the infection is superficial.

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OVEREATING IN LAMBS

(Apoplexy; Enterotoxemia; Indigestion; Gastroenteritis; Food Intoxication; "Pulpy Kidney")

Definition.—Enterotoxemia in lambs is an acute fatal enteritis in highly fed animals caused by *Clostridium welchii (perifringes).*

Etiology.—From Colorado, Newsom and Thorp[1] have reported that it causes more loss in the feed-lots of Colorado than all other causes combined. The mortality sometimes reaches 5 to 10 per cent of the flock and few recover. The practice of "lambing down" (grazing) in fields of corn, barley, or peas has been found to be especially harmful. An increasing prevalence in Colorado has been due to increased feeding of concentrates, such as corn or barley, in order to shorten the fattening period. The feeding period has been cut from 5 months to 90 days, and the difference between a fattening and a killing ration is very slight. Neither alfalfa nor oats alone will cause trouble. The lambs appear to thrive well for a period of two weeks to a month when the disease appears. By this time the leaves from the grain plants and grass from the fence rows have been consumed, leaving mostly grain as the diet. Should high wind blow down the stalks, making more grain available, large losses in lambs are usually encountered the next morning. The cause is attributed to overeating, combined with absorption of toxins of *Clostridium welchii* Type D from the small intestines.[2] That such toxins are responsible for deaths in suckling lambs ("pulpy kidney disease") has been proved by the protective action of *Clostridium welchii,* antitoxin. The disease is occasional in pastured adults. Pulpy kidney disease in lambs, and the isolation of *Cl. welchii* Type D, were first reported in the United States by Boughton and Hardy[3] in 1936.

The contents of the small intestines from lambs dead of overeating are usually toxic for rabbits, guinea pigs, mice, rats, and lambs when given subcutaneously or intravenously. The disease has not been produced experimentally by oral feeding of the toxic material.

Morbid Anatomy.—Decomposition is rapid and if the autopsy is performed three to four hours after death the kidneys are extremely soft (pulpy). There is a hemorrhagic enteritis of the small intestine which may be associated with hemorrhagic gastritis of the abomasum. As described by Miller,[4] the intestinal wall is covered with large subserous hemorrhages, and care should be taken to differentiate between these and the small petechial hemorrhages of hemorrhagic septicemia. There may be subcutaneous, intermuscular, and endocardial hemorrhages. The pericardial sac contains straw-colored serum. In peracute

cases there may be no postmortem changes. The "pulpy kidney" and the friable liver spotted with tawny colored areas 2 to 4 cm. in diameter, best seen when the animal is autopsied three to four hours after death, are considered by Boughton and Hardy[5] to be "almost pathognomonic." Emphasis is placed on distension of the alimentary tract, especially the ileum and the abomasum.

Symptoms.—The attack is sudden with orthotonus, staggering, convulsions, and immediate death, as from apoplexy, or a lamb may be found dead in the morning. More often the course is over a few hours, with orthotonus, walking in circles, and pressing forward. In acute cases the urine contains from 2 to 6 per cent sugar. In less acute attacks there are vomiting, diarrhea, inappetence, and loss of condition, with recovery or death after several days. There may be paralysis with inability to rise for several weeks. Nervous symptoms, hemorrhages on autopsy, and sugar in the urine have not been reported in lambs affected with enterotoxemia in Australia. There is no known remedy.

On the subject of control Boughton[6] writes that "Immunization by injection of a whole culture bacterin (really an anaculture and a toxoid) at the beginning of the feeding period is sound practice. Immunization of the nursing lamb at about one week can be accomplished by the same procedure but is not economically feasible except in small flocks and in valuable breeding ewes and rams. Immunization of the ewe about the end of the breeding season in the fall (October or November), followed by the injection of a 'booster' dose at tagging time (about a month before lambing), is being practiced. The antibodies from the mother are passed into the milk and the nursing lamb may be protected through the danger period.

"In feedlot lambs, starvation for 24 to 48 hours will stop an outbreak. In nursing lambs penning with dams causes an abrupt stop when the lambs should be released in a different pasture. After recovery feed hay sparingly but no grain and return gradually to full feed."

Muth[7] reports that while treatment with antiserum is apparently the most satisfactory method of controlling unsuspected outbreaks of enerotoxemia in lambs 3 or 4 weeks of age, that method is expensive and the immunity is short; in feed-lot lambs, the use of an alum precipitated toxoid appeared to be a promising method of reducing losses. Buddle writes from New Zealand that "the double vaccination of the ewe with *Cl welchii* Type D anaculture has been shown in extensive field trials to be a reliable practical and economical method of control and is now widely practiced in the Dominion."

The use of sulfur in the feed, one fourth ounce per day for hand fed

lambs, and one half ounce for self fed is reported by Deem[9] to reduce the losses, but its value has not yet been generally reported. Methods of control by herd management are described by Newsom.[10]

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INDIGESTION IN EQUINES

There are three chief forms of indigestion in equines: 1, Acute Gastric Dilatation; 2, Intestinal Indigestion with Stasis and Tympany; and 3, Impaction of the Colon and Cecum.

Definition.—The chief characteristics of indigestion in equines are food stasis associated with impaction, fermentation or putrefaction of the alimentary contents, deranged peristalsis and secretion, spasmodic contractions of the muscular walls (colic), as well as various degrees of intoxication and catarrh. While primary indigestion is a functional disorder, it may terminate in inflammatory and other organic changes. Similar functional changes occurring secondary to enteritis, intestinal obstruction and other organic diseases should not be termed indigestion. The great majority of cases are caused by faulty food or improper feeding.

The disease has been described under a variety of names according to some prominent symptom, cause, or condition; thus we have spasmodic colic, engorgement colic, flatulent colic, catarrhal intestinal colic, etc. That an exact classification is difficult is suggested by a report from Behren,[1] that 44 per cent of the colic affections in the Berlin clinic are impactions of the small intestines, and a report from Marek,[2] that 35 per cent of the colics at Budapest are catarrhal intestinal colic. In the classification of indigestion there are two main groups; in one

the location and character of the disease may be recognized by a physical examination, in the other they may not. Conditions that may be recognized are gastric dilatation, impaction—chiefly in the cecum and colon, and tympany. There remains a group, including about 50 per cent of all colic cases, in which an exact anatomical diagnosis is not possible; they are mild indigestions in which the nature and location of the derangement are obscure.

It is probable that in most cases the initial disturbance is in the stomach, since the first effect of overeating is here. In all acute dietetic forms having a sudden onset, one may consider that it is dilated even in the absence of distinct gastric symptoms. Experience shows that often it is not possible to limit the diagnosis to some particular section of the alimentary tract. For example, recently threshed buckwheat straw causes severe indigestion with tympany that involves the stomach, the small intestines, and the colon, while overeating on cornmeal may lead to gastric impaction only. A knowledge of the cause, therefore, is of first importance to a complete understanding of the disease.

Etiology.—*Dietetic Errors* are the chief cause. Foremost is a *change of food,* as from oats to corn, from old grain or roughage to new, and from good to poor quality of rations.

Overeating is responsible for the more serious types of dilatation of the stomach and intestines. This occurs when animals break loose in the night and eat unknown quantities of heavy grain, such as cornmeal, barley, buckwheat, oats, or newly threshed grain. In the harvest field and when tied beside mows, horses obtain new hay, grain, or other roughage to which they are not accustomed. A similar accident follows access to fields of green clover, oats, and other luxuriant crops. Stasis occurs when the bowel is overdistended with gas or food. It sometimes occurs that the morning of the day when the horse is to be given hard work it receives an additional grain ration, causing the indigestion and colic which develop after a few hours.

Indigestible and damaged food are often fatal. In addition to atony and stasis, putrefaction and intoxication are marked, and gastroenteritis may also develop. Horses are less tolerant than bovines to damaged food, such as mouldy silage and hay, filthy bedding, straw and hay picked from the manure in the yard, frozen roots, garbage, soy beans, heated oats or other grains, fibrous grass or swamp hay, buckwheat straw, sorghum, and many others.

Irregular work or standing idle on a full diet is a common history, as frequent as a change of food.

Fatigue.—In the spring months, green horses changed abruptly to increased feed and heavy work are subject to acute indigestions. The

effect of fatigue is a reflex paralysis of the digestive system; a similar effect results from hunger and chilling. Long drives on heavy roads and transportation on the railway are associated with this type.

Drinking excessively directly after severe work and while still warm is a common history, especially when immediately followed by a heavy feed.

Seasonal influence is related to colic through the effect of atmospheric extremes and the presence of indigestible foods peculiar to each time of

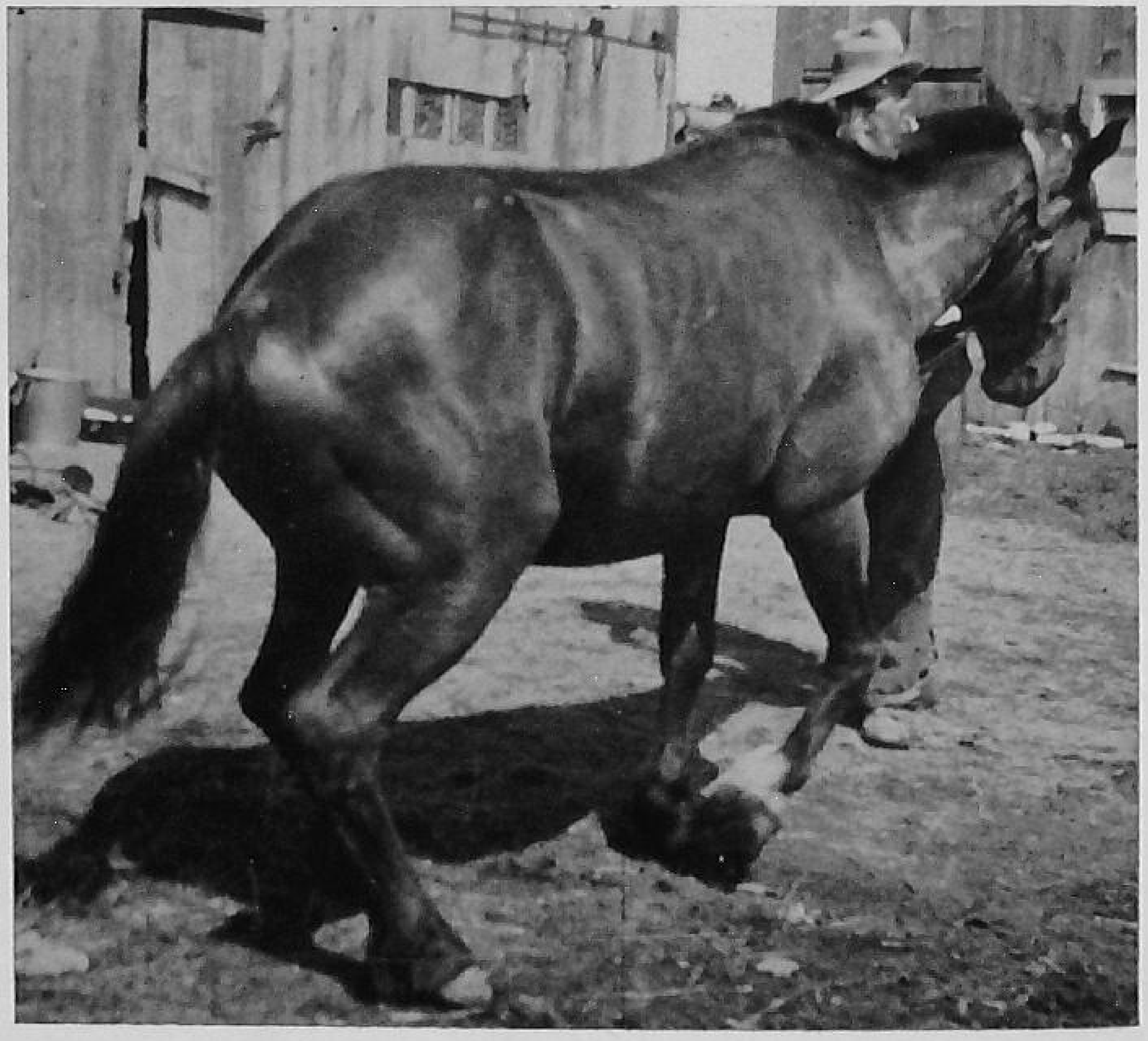

Fig. 15.—Indigestion. (Courtesy of W. J. Gibbons.)

the year. Wall has shown that colic increases with stormy weather. Fatigue and hunger combined with an all-day exposure are often followed by sickness after the evening meal. Increased temperature and humidity are to a lesser degree followed by colic. In the fall horses obtain fermentable foods, in winter fibrous roughage that leads to impaction, and in cold seasons frosted roots and grass. Among farm horses indigestion is rather uniformly distributed throughout the year with an increase in the months of May to August, the period of most active work. There are relatively more impactions of the colon and

cecum in the months of November to April, while gastric dilatation appears to be more prevalent from April to October. In the months of May to August there is a distinct increase in the less severe forms which are diagnosed only as indigestion.

Idiosyncrasy.—There is an occasional horse that cannot eat heavy grain, such as cornmeal, without suffering from frequent acute attacks of colic. In others rapid greedy eating is a cause.

Faulty mastication from defective teeth may lead to habitual indigestion.

Morbid Anatomy.—The mortality from indigestion among horses treated in our clinic has been about 7 per cent, of which slightly more than one half died of gastric rupture. The others had impactions of the cecum, colon, or small colon, usually with rupture; the most frequent location being in the right dorsal colon. In antemortem gastric rupture the tear in the serosa exceeds in length that of the muscular wall and mucous membrane, and the margins of the serosa are hemorrhagic (Fig. 22). In impactions of the terminal portion of the ileum one finds large amounts of fluid in the stomach and small intestines, a comparatively empty colon and cecum, and a hard impacted mass just anterior to the ileocecal valve. There may be enteritis and exudation of serum into the peritoneal cavity.

Symptoms.—In all, there is a history of pain (colic) manifested by restlessness, pawing, and sweating. The onset is usually sudden; it may be directly after eating, when there has been a change of food or an overfeeding; or in the latter part of the forenoon or afternoon, when it may be due to fatigue; or gradual and at any time when the colon is impacted with straw or similar substance. The history often reveals the cause. In severe types the animal rolls and throws itself in a reckless manner as if delirious from pain. Unnatural attitudes may be assumed —dog-sitting position, sternal position with the fore limbs advanced and the body slightly raised; these suggest a dilated stomach. When enteritis, peritonitis, or rupture develop the animal moves cautiously. Stretching occurs in impaction of the colon.

When the trouble is located anterior to the cecum, pain tends to be continuous and severe; when confined to the colon it is often intermittent. Redness of the conjunctival mucosa is an index to the degree of intoxication; in mild forms the color is normal, but in severe and fatal types congestion is marked. An icteric palpebral or scleral mucosa is present in about 15 per cent of cases of impaction of the colon. Often it is present in acute affections of the stomach and small intestines. In both, it is due to catarrh of the duodenum. The pulse and respiration are accelerated in proportion to the severity of the attack. The tem-

perature may be elevated when inflammation and toxemia develop, though shortly before death it is subnormal. When the stomach or small intestines are involved, the course is relatively short; within twenty-four hours the termination is usually apparent. When the colon is the seat of the indigestion, a longer course, extending over two or three days to a week, is not infrequent. The approximate seat and nature of the disease may be recognized by a careful examination of the digestive system, combined with the history, onset, and course.

I. Acute Gastric Dilatation (Acute Gastrectasis.)—Primary gastric dilatation is usually the result of overloading with fermentable or heavy indigestible food, though it may result from stasis alone, without overloading. Gastric dilatation is also secondary to intestinal obstruction, such as stenosis of the pyloric orifice, torsion, or peritoneal adhesions.

Often the owner reports a distinct change of food, accidental access to a field of grain or a grain bin, or that the horse has stood idle for one or two days with no reduction in the food, or has been put to hard work directly after feeding. In most cases the *onset* is sudden within a short time after eating. The pain is severe and continuous. The affection may terminate in one or two hours in recovery; it may end in death in from twelve to twenty-four hours from dilatation or rupture, or in convalescence that requires two or three days for a complete return to normal.

The general symptoms are marked depression, head carried nearly to the floor, profuse sweating, anxious expression, lying on the sternum with the front feet extended and raising the body at each paroxysm, dog-sitting position, red conjunctival mucosa, and fetid breath. The extremities are cool. The animal may dislike to move, and on being forced to walk travels with a stiff cautious gait. At first the horse may throw itself down in a reckless manner and roll and kick violently. After a time it stands quietly or lies down slowly and with care. The pulse is from 70 to 120, small, soft and partly imperceptible; a rate of 100 is a grave sign. The breathing is shallow and fast—30 to 50, and the temperature is from 100° to 103° F., though shortly before death it usually becomes subnormal.

The direct evidence of dilatation is retching, eructation, or vomiting. Wave-like movements pass up and down the esophagus; this is caused by partial eructation of gas that passes nearly to the pharynx and then returns. When vomiting occurs the stomach contents are discharged in small amounts through the nostrils; the ejecta emits a sour odor, and food particles sticking to the nose are evidence that the animal has been vomiting. Passage of the stomach tube may release a reddish sour

liquid, but if the contents are heavy, as in cornmeal impaction, nothing is returned. Rectal examination may cause straining.

The abdomen is bloated slightly or is normal in size; this depends on the kind of food—fermentable or dry. The peristalsis is variable; when sounds are present they are irregular and abnormal in quality; with improvement peristalsis is increased. On rectal examination a change in the position of the spleen is sometimes found; it may be located near the pelvis instead of beneath the posterior border of the costal arch. Often the duodenum is found to be distended where it crosses the median plane behind the trunk of the great mesentery opposite the last rib; this is the usual condition when the food is fermentable—fruit, frozen vegetables, green roughage.

In gastric rupture the animal stands quietly with the head down, bathed in cold sweat, and trembling. The conjunctival mucosa is highly congested, the pulse above 100 and imperceptible, and the temperature subnormal. Peristalsis is entirely absent. Through the rectum one may recognize collapsed intestines occupying the lower part of the abdominal cavity, while the serosa may be roughened by food particles that have escaped from the stomach. A rectal examination may cause pain.

In some cases the overloading is not sufficient to cause distinctive symptoms, and in rare cases these are absent when the dilatation is severe. One should consider the history, the manner of onset, and the exclusion of other types.

Chronic dilatation (gastrectasis) occurs in old animals, where it gives rise to habitual colic that develops when the animal is at work. There are tympany and suppressed peristalsis, vomiting, and expulsion of gas and fetid thin fluid through the stomach tube. Dyspnea with heave-like breathing are brought on by exercise. Regurgitation continues even after the severe pressure has been relieved with a stomach tube. On rectal examination the stomach is found to be enlarged and the spleen is back nearly as far as the brim of the pubis. The prognosis is not good, since such animals are worthless for ordinary work.

II. Intestinal Indigestion with Stasis and Tympany (Catarrhal Intestinal Colic; Spasmodic Colic; Flatulent Colic).—This group includes 70 to 80 per cent of all acute indigestions. The course is brief and generally speaking the symptoms are mild. The condition is rarely fatal, and in such instances one may find enteritis.

The *history* includes most of the causes of indigestion, such as irregular work, change of food, overfeeding, accidental access to feed, eating bedding, spoiled food, frosted roughage or pasture, fatigue, old age, defective teeth, and habitual colic. Regard for the kind and quantity of

food may be the chief means of making a diagnosis. Thus apples, green clover, alfalfa, and access to grain fields induce a gastrointestinal indigestion with tympany and acute catarrh, as well as a very active peristalsis. Exposure to storm and fatigue, on the other hand, are followed by stasis, depressed peristalsis, and more indefinite symptoms.

The *onset* is about equally divided between those that are found sick in the morning, are taken sick while at work, after eating, and after work. In general, it may be inferred that an onset directly after eating is the result of gastric dilatation; this is still more probable if there has been a change of food, a period of idleness, accidental access to food, or feeding of indigestible material. The effect of hard work directly after the stomach is filled is to delay digestion and evacuation, and in these cases the onset may not occur until two or three hours after eating.

The *general symptoms* are often mild, the attitude reflecting the degree of pain. The pain is usually intermittent, so that the animal has spells of rising and lying down, straining as if to urinate, stamping, looking at the flanks, and general restlessness. As a rule the conjunctival mucosae are normal, though icterus is present in a few when it indicates a catarrh of the small intestines. Congestion of the conjunctival mucosa is slight or marked according to the gravity of the case. In about 75 per cent of the cases the pulse rate is between 40 and 65. A rate of 80 or more commonly occurs in tympany. The rate of breathing corresponds with the pulse; in a series of seventy, it was 16 to 20 in 54; 30 to 36 in 12; and 40 to 50 in 3. The higher rates were in cases of tympany. In a series of 200, the temperature was in the nineties in 16.5 per cent, the hundreds in 30 per cent, the hundred-and-ones in 34 per cent, the hundred-and-twos in 14 per cent, and the hundred-and-threes in 5.5 per cent. Temperatures of 103 and above occur chiefly in impactions of the large intestines, and to a lesser degree in gastric dilatation. In either of these conditions the temperature is usually normal, but it may be subnormal. The abdomen is distended in about 10 per cent. The peristalsis is usually active; it may be explosive, irregular, or show a marked increase (borborygmus); it may be suppressed, especially in old animals, and in stasis.

Bowel evacuations are variable—normal, flatus, firm, purging soft watery feces, slight, absent, mucus-covered, sour, or fetid.

In *stasis and tympany of the small intestines* the onset is sudden and the course brief. On *rectal examination* the distended loops may be recognized in the region of the left flank, but in the absence of gaseous distension the intestinal loops may not be felt. In Wall's[3] series of 824 autopsies following colic, 3 per cent were impactions of the small

intestines. While tympanitic conditions of this segment may be recognized, impactions are rarely diagnosed. Autopsies have shown that food accumulation tends to occur in the terminal portion of the ileum. This may possibly be found as a firm cylindrical swelling where the ileum passes towards the base of the cecum at the right of the vertebral column near the transverse plane of the posterior end of the left kidney. Impaction also occurs in the duodenum, which may be found firmly distended directly behind the trunk of the great mesentery. Accumulations of dry residue are rare in the small intestine because of the liquid character of its contents. Fincher[4] has described a case of *impaction of the ileum* observed in our Ambulatory Clinic. A firm mass was located in the terminal portion of the ileum; this caused death within twenty-four hours when a postmortem diagnosis was made. The symptoms were severe and persistent pain, intense congestion of the mucous membranes, occasional dog-sitting position, absence of persistalsis, and a negative rectal examination. In impaction of the terminal portion of the ileum, as described by Harvey,[5] death occurred after a course of about twenty-four hours. The symptoms were severe pain, complete absence of peristalsis and slight bloat. Rectal examination caused straining, and coils of distended small bowel were palpated. Hudson[6] writes that he has had about thirty cases of impaction of the terminal portion of the ileum in a period of three years, that it is the most common cause of death in his practice, and that it is caused by feeding "oat-straw chop, wheat chaff, or rye straw. The chop is usually cut very short, the knives often being set to a quarter of an inch. . . . There is usually a large quantity of fluid in the stomach and small intestines, right up to the obstruction, but it cannot pass it. . . . The symptoms are not very definite."

Stasis and tympany of the large intestines are marked by an increase in the size of the abdomen. On rectal examination one may find the pelvis so filled with tympanitic bowels that the arm is advanced with difficulty. The left segment of the colon may occupy a large part of the left half of the abdomen. Tympany of the cecum may be recognized in the right flank. The bowels contain a pultaceous mass that on evacuation is covered with mucus. Often the mesocolon is found to be tense. In combined overloading and tympany of the cecum and colon, one may at first suspect displacement because of severe general symptoms, congested mucous membranes, absence of peristalsis, and persistent tympany. After the first twenty-four hours, however, it is usually possible to exclude acute obstruction since the condition of the patient does not become hopeless.

III. Impaction of the Colon and Cecum is an accumulation of dry

residue in the large or small colon, less often in the cecum. *Coarse roughage* is the chief cause. In a series of 100 cases treated in the ambulatory clinic at Ithaca, 20 per cent were attributed to straw; then followed poor hay, sorghum, cornstalks, alfalfa, and coarse clover. As in other forms of indigestion, old age, defective teeth, and idleness contribute. About 50 per cent of the cases occurred in the months from October to January.

In one third, the symptoms had been present less then twenty-four hours at the time of the first call. Others had shown constipation two days, partial anorexia from one to three days, while some had been sick from three to five days and even longer. Those with a course of a week or more usually prove to have an impaction of the cecum, or the terminal segment of the colon, and the symptoms are somewhat intermittent. When an owner reports that a horse has been sick with colic for one or two days it nearly always proves to be an impaction of the colon.

The *attitude* indicates less pain than in other forms, the colic being intermittent, but there are wide variations. When due to eating very fibrous roughage the onset is gradual and the pains are at first mild and infrequent. Impactions due to eating recently threshed straw, on the other hand, have a more sudden onset and the pain is continuous. This intermittent colic in combination with irregular persistalsis suggests a so-called spasmodic colic; there is a secondary catarrh of the small intestines. In about 10 per cent of cases of impaction of the colon the horse stands in a stretched-out position, as if to urinate. Tenesmus is somewhat less frequent.

The initial symptoms may resemble those of a mild indigestion of the small intestines, such as intermittent colic and irregular peristalsis. Recovery seems to occur, but on the following day the symptoms reappear in a more severe form. Occasionally the bowel evacuations decrease while the appetite remains unchanged. By the end of twenty-four to forty-eight hours the signs of indigestion are distinct. Inquiry in regard to the diet nearly always reveals a fibrous roughage—"straw colic." The animal is up and down but the actions are not violent. The mucous membranes may be icteric from the secondary catarrh of the duodenum; in the relatively few severe forms, they are congested. The pulse, respiration and temperature are often normal. The pulse exceeds 80 in not more than 10 per cent. With few exceptions a temperature of 103° F. or higher is found in the fatal types, but a high temperature does not preclude recovery.

The *peristalsis* is suppressed, though in the beginning the sounds may be irregular or increased on the left side over the small intestines.

Explosive sounds and increased peristalsis may be associated with mild impactions of the pelvic flexure. The *abdomen* is usually normal in size, sometimes slightly tympanitic. The *evacuations* tend to be suppressed and they may be suspended. The feces are variable—dry, brown and "burned," hard and sour, sticky and massed, or covered with mucus. In the rectum the material is nearly always dry.

The *location of the impaction* is at a place where a segment with a large diameter is terminated by a relatively small opening. Such places are found where the contents of the cecum pass into the colon, where the second segment of the colon terminates in the third at the pelvic flexure, and where the fourth segment unites with the small colon—stomach-like dilatation, transverse colon. *Impaction of the pelvic flexure* and the left ventral colon constitute the rectal findings in the great majority. The condition is easily recognized on rectal examination. Often the pelvic flexure is full and hard and located within the pelvis, where it can hardly be passed without recognition. On palpation of the left ventral colon, the firm mass is easily found. The *terminus of the right dorsal colon* is found to be involved in about 5 per cent and the condition is serious. Marked jaundice and black urine from hemoglobinuria are associated symptoms. The mass may be reached at arm's length in the median plane of the lower half of the abdomen. It is movable and the consistency is like that of a concrement; palpation causes pain. In thin horses it may be possible to recognize the firm movable mass through the right flank. In old horses impaction with dry roughage may occur at the diaphragmatic flexure. In such cases there is complete inappetence, but the general symptoms and the pain may not be especially prominent. Rectal examination causes straining, and death from rupture may occur at any time. *Impaction of the cecum* is in our experience less frequent; on rectal examination, it may possibly be found as a firm mass in the region of the upper right flank. There may be an intermittent course of two weeks or more with a tendency to terminate in rupture. The bowel evacuations are at first diminished or absent, and diarrhea may finally develop even when the impaction persists.

Impaction of the small colon is marked by the presence of a cylindrical, sacculated, firm swelling in the region of the left flank. It is fairly frequent and the mortality is relatively high. It may occur in association with impaction of the terminus of the right dorsal colon. Fatalities are caused by paralysis and rupture of the impacted segment. In one of the author's cases of impaction of the small colon a kink in the rectum caused an incurable stenosis. The course may be a week or more.

Prognosis.—This depends largely on the cause. Buckwheat straw, green alfalfa, and similar roughages cause serious forms. In a series of 125 cases of impaction posterior to the ileocecal valve in our clinic, 21 were fatal. In Wall's[3] series of 824 autopsies following colic, 15 per cent were impactions of the large intestines.

Treatment.—In the treatment of indigestion, the chief objects are to prevent rupture and displacement through control of the pain and tympany, to reestablish normal peristalsis, and to evacuate the bowels.

General Care.—A roomy well-bedded box stall or other similar provision is desirable. When the attacks of pain are frequent and severe a person should be in constant attendance to prevent violent movements and rolling, since these may cause a fatal displacement of the bowel. Our records show that torsion is most frequent in the months when the rate of indigestion is high. Violent movements may be prevented by walking the horse slowly, but rapid exercise is harmful. *Narcotics* effectively control the delirious violence of pain. Chloral hydrate 1 to 2 ounces (30-60 Gm.) is perhaps the best; dissolve in a pint of water and give through a stomach tube. Chloral administered in a capsule may fail to dissolve if the stomach contents are dry. It is also an antiferment.

Dilatation of the Stomach.—A stomach tube promptly relieves gastric dilatation from overloading if the consistency of the contents is not too firm. A tube in common use in America is 3/4 inch in diameter and is passed through the nose; in Europe, a tube of about twice this diameter is passed through the mouth. Evacuated fluids are reddish in color and emit a sour penetrating odor; escape through the tube may be assisted by first introducing a little water. More frequently the gastric contents do not return through the tube; if the material is a heavy substance, such as cornmeal or fibrous roughage, it affords little or no relief. It is convenient and safe, however, for the administration of laxative and other drugs. After a stomach tube has been passed with negative results in gastric tympany, it may be possible to release the gas by means of trocarization of the stomach. The trocar is inserted in the 17th intercostal space at the level of the tuber coxi, and passed in the direction of the right elbow. For the control of tympany one may give creolin, turpentine, or soluble pine oil 1 ounce (30 Gm.) in a pint of water; other useful carminatives are aromatic spirits of ammonia 1 to 2 ounces (30-60 Gm.), ammonium carbonate 4 to 8 drams (15-30 Gm.), capsicum, ginger, and sodium bicarbonate 1 to 2 ounces (30-60 Gm.). Alcohol 1 to 2 ounces (30-60 cc.) in a quart of water is recommended by Ferguson.[7] The value of alcohol for relief from the discomfort of an overloaded stomach has long been recognized.

Salicylic acid 4 to 6 drams (15-25 Gm.) is also widely used to control gastric fermentation.

Laxatives that soften the mass are indicated; among these are mineral oil 1 gallon (4 liters), raw linseed oil 1 quart (1000 cc.), aloin 4 drams (5 Gm.), or sulfate of magnesia (¾ to 1 lb.). These are combined with carminatives and antiferments. When there is known to be severe overloading, as from coarse roughage, the gallon or more of mineral oil may be supplemented by a quart of raw linseed oil, or a pound of sulfate of magnesia, or 2 drams of aloin, and the mineral oil may be repeated at the end of 12 to 24 hours. The problem is to evacuate the digestive tract and to prevent rupture of the stomach or intestines. In severe gastric dilatation the immediate addition of bulky material may be harmful. One must adjust the treatment to the condition and avoid a routine treatment. It may be necessary to keep a catheter in the stomach for one or two hours at a time and to trocarize the cecum. Two ounces each of alcohol and chloral in a quart of water are often beneficial. Fluid extract of nux vomica 2 to 4 drams (8-15 cc.), or strychnine sulfate ¼ to ½ grain (0.015-0.03 Gm.) two to three times daily are frequently given for the stimulant effect upon the digestive system. Arecoline hydrobromide ¼ to ½ grain (0.015-0.03 Gm.) has been widely used in a routine manner in all forms of colic; it is of doubtful value in gastric dilatation since it may contribute to paresis and rupture of the stomach wall.

Intestinal Tympany and Stasis.—When intestinal tympany is distinct the bowels should be trocarized. This is without danger to the animal, if a small, sharp, clean instrument is used. The pain and tension that suspend the physiological action are relieved. The most frequent location for the relief of tympany is over the cecum which is trocarized through the right flank. If this is not successful the left flank is trocarized, releasing gas directly from the colon. The operation may be done repeatedly on both sides until tympany is relieved. When the ingesta is a seething mass of green roughage and gas, a solution of creolin (2 per cent) or other antiferment may be introduced through the canula. Trocarization of distended intestinal loops may also be safely done through the wall of the rectum; for this purpose a 16 gauge 1¼-inch hypodermic needle with a large hub is suitable.

Laxatives and carminatives are indicated as in gastric dilatation. When distension is not too great, peristalsis may be stimulated with arecoline hydrobromide ¼ to ½ grain (0.015-0.03 Gm.) repeated once or twice at half-hour intervals. In mild forms it is of doubtful value to administer an aloin or other purge that of itself may keep the animal from service for two or three days; one may give a carminative,

arecoline ½ grain (0.03 Gm.) and prescribe a mixture of equal parts soluble pine oil, mineral oil, and turpentine 2 ounces (60 cc.) every half hour.

Lentin (2-4 cc.) subcutaneously has been introduced as a substitute for arecoline. It acts directly and immediately through the vagus. On the digestive tract it causes increased glandular secretions and increased peristalsis. It slows the pulse and increases its strength. It is contraindicated in weak subjects and in overloading of the stomach. Atropine neutralizes the effect. While it is recommended to be given subcutaneously, one half of the dose of 4 cc. may be given in the vein and the other half subcutaneously; when given in this manner evacuation of the bowels often follows immediately. It is no safer than arecoline.

In more severe types, marked by fast pulse, severe pain, congested mucosae, and suppressed peristalsis, a strong laxative is indicated—aloin or oil. Or one may give a gallon of mineral oil containing 1 ounce (30 cc.) each of alcohol and chloral hydrate, and follow with atropine sulfate ½ grain (0.015 Gm.). If the evacuations are still depressed at the end of 24 or 48 hours, one may give a pound of salts dissolved in a gallon of water. While it is a common practice to give arecoline in such cases, its value here is questionable. After the administration of arecoline an increased pulse, increased pain, and weakness, suggest an injury, possibly a displacement or rupture.

Impaction of the colon is relieved by the use of laxatives. When the general symptoms are not marked, administration of oil may be followed in ten or twelve hours with arecoline hydrobromide ¼ to ½ grain (0.015-0.03 Gm.). In aged horses and in firm impaction it is better to omit arecoline and administer two quarts of mineral oil twice daily until the impacted material is softened and expelled. This may require three or four days, though the average impaction responds within forty-eight hours. The chief danger is from rupture of the intestine. The secondary catarrh of the small bowel is controlled with creolin 4 to 8 drams (15-30 cc.). Sodium or magnesium sulfate 1 lb. (500 Gm.) in a gallon (4 liters) of water administered through a stomach tube is also useful in impaction of the colon.

Enemas of warm water have long been used in the treatment of impaction in the horse. As commonly used, they are of doubtful value except in accumulations in the rectum or posterior part of the small colon. Various syringes have been devised that block the return flow of the water. Two or three gallons or more are introduced on the theory that it reaches the colon and lubricates or softens the impacted mass. They are of undoubted value in certain cases. One of the most fre-

quent seats of fatal impaction is the terminal end of the right dorsal colon, sometimes extending over into the small colon; in this form a special rectal syringe is useful.

Treatment of impaction of the cecum with intravenous injection of Lang's solution (30 Gm. sodium citrate, 30 Gm. sodium chloride, 500 cc. sterile water), as recommended by W. W. Lang[8] of Great Britain, has been used. The action is to increase the thirst, causing the animal to drink large volumes of water and thus soften the impacted mass. The injection should be made slowly. It is doubtful that this will replace the use of mineral oil or other laxatives in the relief of severe impactions, but it may be a useful adjunct.

Gratzl[9] writes that the following treatment for relief of impaction of the colon has been found effective in the Vienna clinic: liquid petrolatum 2 to 3 quarts (2-3 liters), often repeated once or twice, and 14 quarts (14 liters) of warm water daily through the stomach tube. The patient also receives atropine sulfate ¾ to 1½ grains (0.045 to 0.09 Gm.) daily.

For relief of impaction of the small colon, Carlisle[10] writes that the only solution is to break the mass down by manipulation. Pass the hand over or under the portion of the colon containing the mass and float it back into, or to, the pelvis. Here the mass is massaged against the rim or floor of the pelvis until broken into pieces. This may require an hour or more.

Depression and weakness with impending collapse in any form of indigestion may be combated with large amounts of normal saline solution (2500 to 5000 cc.) in combination with 40 per cent dextrose solution (500 cc.) and 20 per cent calicum gluconate solution (500 cc.) per vein. For an attack of laminitis associated with indigestion, place ice packs around the hoofs during the first 24 hours, and each day thereafter force the animal to walk a short distance three or four times. When the season permits, a horse with laminitis should be turned loose in a paddock or pasture.

Prophylaxis.—When a horse has been exposed to rain, wind or exertion, and is hungry, he should be rubbed dry and given not more than 4 quarts of water; a little hay may then be fed, followed in an hour by 4 to 5 pounds of grain.

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CONSTIPATION

Constipation is defined as delay in the passage of the contents of the intestines. As a primary affection it is caused by coarse indigestible roughage. Some cases are closely related to subacute or chronic indigestion, while others are more of the nature of acute catarrh. According to the age and species there are several distinct clinical types. It is met with frequently in *calves* when the change from milk to roughage has been too abrupt, or when the hay has been too fibrous. The ill-effects of fibrous material in the stomach of the young calf are described in the section on diseases of the newborn. Between this condition and constipation, or constipation alternating with diarrhea, in slightly older calves, there is no distinct line. The individual is unthrifty, loses weight, grows long hair, and often has a perverted appetite. Even when suitable roughage is offered, a preference is shown for coarse stalks, and filthy bedding. Laxatives of oil afford only temporary relief if the animal still has access to faulty food. The course is indefinite. Frequently nothing more definite is recognized than a calf that, for some unknown reason, failed to thrive. Unthriftiness and loss in condition may lead to emaciation and result in a badly stunted animal. Or diarrhea may alternate with constipation and lead to death from exhaustion. The best remedy for calves that are still on milk, or that have recently been weaned, is to apply a muzzle to prevent the eating of roughage, and resume a milk diet. A calf 4 to 6 weeks of age may improve rapidly when placed on a nurse cow. Laxatives, according to the needs, and bitters are useful. This trouble may be easily prevented by supplying a suitable quality of hay for calves. A year's supply of mixed hay, containing some clover, and harvested in June, is ideal.

A similar condition exists in *yearlings* and young stock, especially in the early winter, when they change from pasture to a diet of coarse overripe and overcured hay. It may be caused by the deliberate eating

of indigestible substances, such as shavings or buckwheat hulls used for bedding. An animal may even eat shavings and sawdust, as it would grain. The symptoms are characteristic. The animal is usually below the others in size; the hair is long; the head big, and the general appearance poor. Constipation is marked and persistent, the feces being black, hard, and scanty. After the condition is well-established, it persists even after a suitable diet is provided. The treatment is to provide an abundance of water and succulent food, daily exercise, mineral oil in sufficient quantities to produce a laxative action, and bitters. The combination of Carlsbad salts, nux, and gentian, as prescribed for indigestion, is useful here. Addition of molasses to the diet may result in improvement.

Horses upon a fibrous diet of coarse hay or clover, especially when overripe and overcured, may developed anorexia and lessened bowel evacuations. Impaction of the colon is suspected but a rectal examination is negative; the animal is merely constipated. The feces are dark-brown, "burned," pasty, and perhaps covered with mucus. There may be jaundice. The abdomen is gaunt and the peristalsis weak or irregular. A similar condition in *cows* is met with less often; the milk flow and bowel evacuations gradually diminish, and there may be acetonemia. In the horse, debility from old age or overwork, faulty mastication from defective teeth, or parasitism lead to an unthrifty condition and dullness; the feces are dry and brown and mixed with indigestible food or mucus. In constipation in adults, discovery and removal of the cause, followed by a course of bitters, brings prompt improvement.

INTESTINAL OBSTRUCTION

Intestinal obstruction is any hindrance to the passage of the feces. It may result from displacement of the stomach or intestines, an obstruction or compression stenosis, or a stricture. No sharp line can be drawn between obstructions due to food residue and those arising from other causes; the former are described under indigestions.

VOLVULUS

(Torsion; Gut-Tie)

Volvulus is an *acute obstruction* caused by rotation of a segment of intestine around its mesenteric axis, its own longitudinal or transverse axis, or an axis composed of another intestinal coil and its mesentery. This is the most frequent form of intestinal obstruction in equines, and it is infrequent in other species. Statistics indicate that in colic in the horse, 55 per cent of the mortality and 5 per cent of the morbidity are due to torsion. In a series of 715 colic affections treated in our ambula-

tory clinic, torsion comprised 6.7 per cent of the sick, and 40.7 per cent of the autopsies; 40 per cent affected the colon, and 60 per cent the small intestines. Volvulus of the small intestines is either mesenteric, or a rotation of one segment around another intestinal coil; the ileum with its long mesentery is the most frequent location of the twist. Volvulus of the colon is usually a rotation around its own longitudinal axis at the diaphragmatic flexure. Rare types are rotation of the colon at the base of the cecum, even involving the latter; rotation of the left ventral and dorsal colons around the cecum; kinking of the cecum near the tip, kinking of the small colon, or kinking of the colon near the pelvic flexure; and torsion of the stomach in bovines, especially in calves.

Etiology.—*Indigestion* associated with colic is the most frequent cause. Irregular peristalsis and unequal filling of the bowel, combined with rolling and violent movements account for the majority. In the writer's series, indigestions are most frequent in October and November, and in these months are found 40 per cent of torsions. The primary disease may be a dilatation of the stomach, an impaction of the colon, or a digestive trouble extending over two or three days. Others have a history of habitual colic, have had free access to cold water when heated, or have been exposed to one or more of the various causes of indigestion.

Mechanical displacements, independent of indigestion, also occur. These are observed in horses that struggle when cast or that have rolled over an embankment; an immediate onset leaves no doubt as to the cause. It is commonly stated that maneuvers of army horses render them especially susceptible to torsions.

Predisposing factors which explain its frequency in the horse are found in the long mesentery of the ileum, and the free portion of the colon.

Morbid Anatomy.—Torsion causes more or less transudation of reddish serum into the abdominal cavity, the amount depending on the length of intestine involved in the twist. All parts distal to the compression are distended and dark, the wall thickened, and the contents stained with dark-colored serum. Occasionally a slight twist may disentangle as the bowels are removed from the abdomen, when the condition is recognized by the presence of a dark segment sharply defined from the normal intestine.

Symptoms.—The onset is sudden and is marked by severe and continuous pain. Often the horse is found in the morning, covered with bruises and inclined to remain down. A history of violent exercise, falling, or being cast is suggestive. Generally speaking the immediate

history is variable. At first the pain may seem to be mild, but more often one finds dejection, anxiety, distress, delirium, sweating, and violent movements. The latter may soon give way to a quiet, recum-

Fig. 16. Torsion of the colon and cecum. A, marked distension of the colic vessels, recognized on rectal examination as a cord-like painful mass at the pelvic flexure; B. D, C, stomach, small intestines, and small colon, each of which is normal. The dark-colored cecum and colon were included in the torsion.

bent position. When standing, pain is manifested by pawing, kicking the belly, turning the nose to the flank, and moving about. The head may hang nearly to the floor and the ears droop, as in gastric dilatation. A stretched out position, as shown in Fig. 17, is not infrequent. From time to time, the animal sinks almost to the floor in a crouched

position as if to lie down and then slowly rises again. A few assume a dog-sitting position. Towards the end the pain lessens and the owner anticipates recovery. Congestion of the mucosae of the eyelids gradually develops until it becomes marked and diffuse. The breathing is labored and variable in frequency. The pulse at first is 50 to 70, then it gradually increases to 100 or more and becomes poor in quality. The temperature may be 103° to 105° F. at first and gradually fall to subnormal; it may remain up until the end or it may gradually rise. More

Fig. 17.—Torsion of the small intestine. Note dejected expression, drooping ears, and dilated nostrils. (Courtesy W. J. Gibbons.)

often it is 99° to 102° F. A rising pulse and temperature are of grave significance. Trembling is frequent.

Anorexia for food and water is complete. When the small intestines are affected, vomiting or retching may occur, and the abdomen is either normal in size or slightly bloated. In torsion of the colon persistent bloating is the rule. Bowel evacuations are almost entirely suppressed. Peristalsis is absent or slight; it may be limited to an occasional gurgle or tympanitic drop. Tenesmus is frequent and in our series it has been limited almost entirely to cases of torsion of the small intestine. The animal usually strains when a rectal examination is being made.

A *rectal examination* may be the only means of making a positive diagnosis. In torsion of the small intestine, rectal examination in the upper left part of the abdomen may reveal an irregular tympany:

or at some particular area one may locate a painful spot that when touched causes the animal to groan and kick the belly. Loops of the small bowel may be found displaced to the right above the colon and towards the cecum; a loop of small intestine may show increased consistency, as well as pain to the touch. Mesenteric volvulus of the ileum may present a tense cord-like twist of the mesentery in the region of the left kidney, but a tense condition of the mesentery is also found in primary indigestion. In torsion of the *colon,* tympany is usually marked; trocarization does not relieve the pain, and bloating soon returns. Anterior to the pelvic brim one many find a cord-like painful mass (see Fig. 16A) composed of the colic vessels. A distinct prominence of the longitudinal bands is suggestive of torsion of the colon. Ability to diagnose displacements of the intestines by means of a rectal examination requires much practice. Regardless of what may be felt on rectal examination, a diagnosis of displacement or any other form of acute intestinal obstruction should not be made unless it is supported by the general and digestive symptoms which invariably characterize such obstructions: continuous pain, absence of peristalsis, absence of bowel evacuations, intense congestion of the conjunctival mucosa, pulse fast and of poor quality.

The *course* is from eight to twenty-four hours, terminating in death. In slight torsions of the colon, the course may be prolonged to forty-eight hours or more. There is no cure.

INCARCERATION

(Strangulation)

This is an *acute obstruction* that occurs when a loop of intestine passes through a natural or artificial opening in the peritoneum and is held there, or when the bowel is strangulated by the long stem of a pedunculated tumor or by other fibrous cords or bands. Statistics indicate that in equines it constitutes about 1 per cent of the morbidity and 6 per cent of the mortality from colic; in our series the rates are about 2 per cent and 12 per cent. It is not infrequent in cattle. In young foals the bowel may be compressed by the urachus; in cows, a segment of intestine may be suspended over a fibrous adhesion between the rumen and uterus, or over other bands or cords that develop in chronic peritonitis. In stallions, incarceration in the inguinal ring, due to inguinal hernia, is not rare, and this possibility should be considered when one is examining such an animal affected with colic. In bovines the most frequent locations are peritoneal adhesions; in equines, pedunculated tumors and slits through the mesentery and omentum are the

chief cause. A segment of intestine may also pass through a slit in the diaphragm. Rolling and tumbling from colic seem to predispose to the disease.

Symptoms.—In equines, the general symptoms are like those of torsion; in bovines, like those of intussusception. On rectal examination, it may be possible to recognize peritoneal adhesions in the cow, and inguinal incarceration in the horse. With these exceptions, differentiation from other forms of acute intestinal obstruction is hardly possible. The possibility of an exact anatomical diagnosis is greater in the cow, where, in suspected obstruction, one may explore directly through an incision in the flank or the vaginal roof.

Treatment.—Inguinal hernia in the horse may be relieved by an early operation; in a few hours incarcerations become necrotic and incurable. In cows, on the other hand, compression of the intestines may be successfully relieved by means of an operation even after the condition has existed for two or three days.

INTUSSUSCEPTION

(Invagination)

This is a form of acute intestinal obstruction caused by the telescoping of a section of the bowel into a portion immediately behind it. The affected part forms a sausage-shaped, firm, painful swelling comprised of three segments: an outer or ensheathing, a middle, and an innermost layer. It occurs chiefly in cattle, is not uncommon in sheep, and is rare in the horse. About one case a year is met with in cows in our ambulatory clinic. The *etiology* is explained by an irregular peristalsis whereby a portion under contraction slips into and is overlapped by a relaxed portion. Tumors of the lumen of the bowel are also a cause; these are seized by peristaltic contractions and dragged into the adjoining segment. Bosshart[1] reports that in 36 operated cases he has always found some form of tumor or inflammatory growth in the lumen of the affected part. In a series of 23, 15 occurred in the months from January to June, 5 were in March, and 3 in January.

Symptoms.—The onset is sudden and marked by complete anorexia and colic. The cow may twist the tail, tread constantly, stand stretched out with the feet resting behind the gutter, or remain down. She is usually depressed, sometimes alert. After twenty-four to forty-eight hours the pain recedes. The visible mucosae show little change. The extremities are cool; chills are frequent and often marked by quivering in the flank. The pulse ranges from 90 to 100; it may be as low as 70 or as high as 130. In all of our cases the temperature has been normal, with the exception of one that carried 104° F. for a time but

soon dropped to 100°. The breathing is 18 to 30, shallow, irregular, and catchy. The peristalsis is entirely suppressed and the abdomen is about normal in size. Palpation in the right flank over the region of the small intestine is often painful. The bowel evacuations are in most cases entirely suppressed; one notes a tar-like bloody discharge. In rare cases, the patient continues to discharge a small quantity of fetid feces. *Rectal examination* nearly always reveals a firm, painful circumscribed swollen gut anterior to the brim of the pubis in the right flank; this may crepitate. The small intestines are distended with gas. According to Bosshart,[1] the empty rectum contains a distinctly characteristic exudate in the form of a sticky, mucilaginous substance. Exudates that will not stretch or that retract into a solid mass indicate croupous enteritis. The *course* of intussusception in cattle is from six to eight days. Recovery is possible when relief by operation is delayed even to the fourth day. *Treatment* consists in resection of the affected part and anastomosis of the normal ends.

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STRICTURE AND FOREIGN BODIES

These are infrequent causes of *acute and chronic* intestinal obstruction due to changes in the wall (stricture), tumors, foreign bodies, parasites, cysts, abscesses, and other conditions that either compress or occlude the intestine. Chronic bloat caused by masses of lymphoid tumors involving the forestomachs and by papilloma of the reticulum have been described.[4,5] In young calves, a circumscribed inflammatory growth at the pyloric orifice of the abomasum (pyloric stenosis) occurs rarely as a cause of tympany after eating, and it may prove fatal. The larval form of *Oestrus equi* (bots) may occlude the pyloric orifice of the stomach or the first few inches of the duodenum, causing acute obstruction and rupture. We have had two of these cases in the month of June; the symptoms were like those of gastric dilatation. In circumscribed peritoneal adhesion between a small area on the small intestine and the parietal peritoneum, there is a history of frequently repeated severe colic. In this condition there is a kink in the small intestine which causes partial obstruction. Finally acute inflammation and swelling of the involved parts cause complete obstruction. In the final attack the course may be from one to three days. In obstruction of the small intestine the termination is usually reached in approximately twenty-four hours; in circumscribed adhesion, the development of the obstruction is gradual and the attack may be prolonged for from two

to three days. Abscess formation after infection from trocarization, or from irregular strangles, may gradually compress the intestines of equines, and a similar condition may develop in cattle affected with traumatic gastritis. The rectum may be compressed with melanotic tumors, abscesses, or cysts. An impacted small colon led to a ventral kink in the rectum, and death from acute obstruction.

Sand colic is a special form of impaction of the colon caused by the ingestion of large quantities of sand while eating off the ground. It occurs chiefly among army horses fed in paddocks or in the picket line. Wright[2] has described a case in a mule which deliberately ate sand.

Concrements (Enteroliths) are rare. They are usually found in the terminal part of the right dorsal colon of solipeds. Only the larger ones cause obstruction. They consist largely of the phosphates of lime and magnesia. The etiology is attributed to the feeding of bran or other substance rich in phosphates. In the museum of the New York State Veterinary College is a 10-pound calculus that caused the death of a horse used in a lime quarry. Pieces of nails or other metal in the feed seem to predispose; the center or nucleus is often formed by a one-half inch piece of nail. It is probable that the salts in the food or water of certain localities account for the marked geographical variation in the distribution of this disease.

Symptoms.—These vary widely according to the seat and nature of the cause. Obstructions affecting the stomach or small intestines are either acute or become so after a few attacks of colic, as in the case of tumors (cancer of the small intestine) or circumscribed peritoneal adhesions. The final symptoms resemble those of torsion, but the course may be somewhat longer. In pyloric stenosis in calves, the condition is poor and bloating occurs after each meal; it is fatal in from two to four weeks. In multiple abscess formation after trocarization or strangles in the horse, there is a gradual loss in condition. On rectal examination the hand meets with obstruction and it may be impossible to recognize the usual landmarks. Similar conditions occur in cattle as a result of traumatic gastritis or perimetritis. Compression of the rectum from a tumor, cyst, or abscess leads to a mild attack of colic associated with constipation. The obstruction is recognized by means of a rectal examination. In case of doubt as to the character of the swelling, an exploratory puncture is indicated. With the formation of a large cyst which may project into the lumen of the small colon, there finally develops sufficient obstruction to cause colic. Since the obstruction is not complete the symptoms are not intense but they are persistent. On rectal examination the hand meets with a circumscribed soft mass that may seem to be from 6 to 8 inches in diameter. On palpat-

ing the cyst through the wall of the rectum, one gets the impression that it is a distended mass of displaced intestine, as in a torsion or an incarceration, but the mild and intermittent character of the colic rules out the diagnosis of an acute obstruction. A definite diagnosis may be made by puncture of the swelling with a 16-gauge hypodermic needle. The contents may be collected through a small rubber tube attached to the hub of the needle.

Sand colic is characterized by a sudden onset leading to severe and continued pain. The animal rolls, assumes a dog-sitting position, and often throws itself in a reckless manner; in this way the colon may be ruptured. The pulse and respiration are increased but the temperature is normal. The abdomen is small and peristalsis is suppressed. Bowel evacuations may cease or the animal may pass thin feces heavily mixed with sand. Lieut. Dean[3] reports that a sand impaction of the diaphragmatic flexure commonly develops in from ten to twelve days in horses that have access to grain spilled on a sandy picket line, that the feces may be 95 per cent sand, and that greedy animals are most susceptible.

The symptoms of *concrement obstruction* are those of habitual severe impaction of the colon. A positive diagnosis is made by finding the stone in the terminal end of the right dorsal colon, but it is difficult to distinguish between a firmly impacted mass of food and a concrement.

Treatment.—In the *horse*, a perirectal abscess or cyst is brought to a termination by thorough drainage. Tumors may be successfully removed if they are not too extensive. In sand colic and obstruction from a concrement, administer chloral to control the pain and give large doses of mineral oil (1 gallon); the mortality is high. In *cattle*, chronic obstructions are so extensive that they are rarely operable.

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GASTROINTESTINAL CATARRH

(Intestinal Catarrh; Gastric Catarrh; Diarrhea)

Gastrointestinal catarrh is characterized by hyperemia and swelling of the mucosa, an increase of mucus, and diarrhea, though the last may be absent. It may be limited to the stomach or an area in the intestines; usually both are involved.

Etiology.—In mature animals the chief cause of *primary catarrh* is a faulty diet, such as food that is frozen, green or mouldy, and abrupt change from old roughage or grain to new, or any of the causes of indigestion. Other causes are exposure to cold, overwork when green, irregular work, fatigue, old age, defective teeth, and infection (winter dysentery, Johne's disease, pyelonephritis, hemorrhagic septicemia, coccidiosis). In impaction of the cecum diarrhea may be present from the beginning. Buckwheat straw or ground buckwheat in the grain may cause diarrhea as a symptom of intestinal indigestion. Feeding from a recently opened silo, especially in the fall, may result in an outbreak of scours in cows and a drop in the milk production. In individual cases dysentery may persist for several days after the general recovery in the herd. Certain "high-strung" horses have diarrhea when worked or driven; this occurs in race horses and is commonly attributed to a nervous temperament.

Intestinal catarrh is *secondary* to acute general infections, such as septic metritis, and is a *partial condition* in most affections of the digestive system. Chronic diarrhea is a common symptom of strongylosis in horses, stomach-worm disease and nodular disease in sheep, and ascariasis in swine and horses. In a herd of dairy cattle, diarrhea may persist in a cow for weeks from no apparent cause. Shipping dysentery occurs in feedlot lambs, where it may be associated with coccidiosis and also combined with pneumonia.

Symptoms.—Anorexia, lessened milk flow in cows, and dullness are usually present. The conjunctival mucosa may be icteric or congested, but in most cases of primary bowel catarrh it is normal. Fever may be present, especially in calves. Diarrhea is usually present, though in mild forms confined to the small intestine it may be slight or absent. It may alternate with constipation and the bowel evacuations are often fetid. *Fermentable food* causes a diarrhea that persists for several days. Peristalsis is at first increased and loud rumbling sounds may be heard at a distance (borborygmi). A severe type that tends to develop into gastroenteritis occurs in cows after they have eaten mouldy or frozen silage or cornstalks, or decomposed roots. Mouldy silage from the top of a recently opened silo shortly after it has been filled, in the fall, seems to be especially dangerous. Mouldy or frozen silage taken from the lower layers in the winter or spring is usually harmless to cattle.

Diagnosis.—It is of first importance to differentiate between a primary catarrh and one that is secondary or partial. By some the term catarrh is applied to indigestions, as catarrhal intestinal colic in the

horse (Marek), and in certain types either name may be justified. In its typical form, however, indigestion is more intense and painful, runs a shorter course, and is not associated with diarrhea. Gastroenteritis shows greater depression, more general disturbance, and a less active peristalsis.

Treatment.—Remove the cause, give rest, and provide warm dry quarters. When due to a faulty diet, laxatives are indicated unless evacuations have already occurred: liquid petrolatum 2 to 4 quarts (1-2 liters) when there is irritation from fermentable or decayed food. A laxative and a change of diet are sufficient in mild attacks, though it may be beneficial to follow with a short course of bitters, such as strychnine sulfate ½ to 1 grain (0.03-0.06 Gm.) orally thrice daily, or:

℞ Sal Carolini factitii	℥ xvi (500 Gm.)
Genitanae	
Nucis vomicae	aa ℥ viii (250 Gm.)

M. Sig. Tablespoonful (15 Gm.) thrice daily for a horse or cow.

When evacuation has been accomplished, either by laxatives or by natural means, and diarrhea still persists as evidence of a continued irritation, intestinal antiseptics and protectives are useful: creolin 4 to 8 drams (15-30 cc.) is especially indicated in infectious types (winter dysentery); equal parts of pine oil and creolin, two #10 capsules (40 cc.) two to three times per day; zinc sulfocarbolate 1 to 4 drams (4-15 Gm.); salicylic acid 1 to 2 ounces (30-60 Gm.); tannic acid 2 to 8 drams (8-30 Gm.); or a 4 per cent solution of chlorine (60 to 120 cc.). The following diarrhea mixture is widely used: sodium bicarbonate, 45% catechu, 45%; zinc phenolsulfonate, 10%. Among the sulfonamide derivatives, a limited use of sulfathaladine suggests that it is highly useful in the control of diarrhea in cows. An effective dose is 8-15 Gm. per 100 pounds (50 kg.) body weight daily. In irritation of the mucosa, bismuth subnitrate 2 to 4 ounces (60-120 Gm.) daily is effective. In tympany and acid fermentation, antiferments are indicated: sodium bicarbonate 1 to 2 ounces (30-60 Gm.); ammonium carbonate 1 to 2 drams (4-8 Gm.); or aromatic spirits of ammonia. In the nervous diarrhea of race horses, Dover's powder 4 to 8 drams (15-30 Gm.) is recommended. Opium and its preparations are generally advised to check excessive exudation and peristalsis after evacuation. In selected cases opium may be of great value, but it is too expensive for routine use. When there is weakness after persistent diarrhea, calcium gluconate (500 cc. of a 20 per cent solution) and dextrose (500 cc. of a 40 per cent solution) given intravenously are beneficial.

For dysentery in lambs Forsyth[1] recommends sulfaquinoxalin in

which one pint of a 3.2 per cent suspension is added to 15 gallons of drinking water.

Croupous Enteritis

Croupous enteritis is a mild superficial type affecting chiefly the large intestine and characterized by diarrhea with the presence of croupous or fibrinous shreds, membranes, or casts in the feces. It is most frequent in cattle in the cool weather of the spring when luxuriant grass may be frosted; it may follow the feeding of rotten potatoes, or a sudden change to green roughage. It rarely occurs in the horse and sheep.

Symptoms.—There is a fetid diarrhea containing croupous membranes and blood; the membranes may be tube-shaped casts of the colon and they have been mistaken for a sloughed section of the intestine. The symptoms in cattle are those of indigestion with colic and tenesmus, and a diminished milk flow. The colic is relieved in a few hours, the entire course in about a week, and recovery is the rule. For treatment, Epsom salt is especially recommended.

Reference

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WINTER DYSENTERY

(Infectious Diarrhea of Cattle; Vibrionic Enteritis of Calves)

Winter dysentery is an enzootic diarrhea in stabled cattle occurring in the winter and early spring. Usually it is mild and transient and does not pass beyond the stage of acute bowel catarrh, but it may be severe. The cause has been attributed by Jones and Little[1] to vibrio which they named *Vibrio jejuni*, n.sp., because of its chief habitat in the jejunum.

Etiology.—Veterinary literature does not indicate the prevalence of this disease, but it is frequent in the northeastern part of the United States from New England to Ohio; it has been described by Steffen,[2] and Boyd[3] in the Middle West, and probably it has a much wider distribution. In recent reports of the Ambulatory Clinic of the New York State Veterinary College treatment of 200 to 300 cases annually has been recorded. An acute usually non-fatal type of diarrhea that appears to be spreading has been observed for a number of years in the fall and winter in southern England—Rollinson.[4] Examinations have not revealed the cause, but apparently the disease in England is identical with that described by Jones and Little.[1] While it may be confined to a given area, it may also have a more haphazard distribu-

tion with no explanation of the source of infection; it may spread along the road from farm to farm as if carried by persons or animals; or the herd may come down with dysentery on about the third day after it is left in the care of a neighbor who comes from an infected stable. Occasionally an outbreak occurs after the owner has visited a neighbor's sick herd, and a claim for damages from a suspected human carrier has been the cause of expensive litigation.

In the reports by Jones and Little,[1] they describe experimental transmission of diarrhea which appears in calves three days after feces or intestinal contents or cultures of vibrios of diarrhea were fed by mouth. Cultures of vibrio were obtained from the small intestine, especially the jejunum, and organisms of similar morphology were found in preparations of mucus obtained from the feces of cows with diarrhea. Pure cultures were obtained when bits of ground mucosa from the jejunum were first inoculated into condensation fluid of slanted agar to which a few drops of horse blood had been added and after reinoculation of the condensation fluid into the secondary tubes. "When autoclaved moist feces were inoculated with vibrios and stored in the room the vibrios lived for six days, but when the feces became thoroughly dried cultures could no longer be obtained."[5] Like the vibrio of Asiatic cholera, survival outside the body is brief. Cultures soon die when dried in the air. At a temperature of 55° C., and when exposed to 0.5 per cent phenol, vibrios die in a few minutes. They form no spores and the reservoir of infection between seasonal epidemics is unknown, but probably it is similar to that of the Asiatic cholera in man which may be contracted from recently recovered patients or from those who are in the incubative stage or from healthy carriers who may become infected and excrete the vibrios without manifesting any sign of the disease and who may finally develop cholera.

Attempts to reproduce winter dysentery experimentally by drenching cows with feces from the sick are usually negative; this may be due in part to the time interval between collection and administration of the sample. From several attempts to reproduce the disease by drenching a cow with feces from the sick, I have been successful only once, and in this case the infection was also transmitted from the experimental cow to her calf. Similar negative results have been experienced by students of Asiatic cholera, where negative experiments, as Koch[6] pointed out, are of little value. "We do not know all the factors that are necessary for the reproduction of the disease, and quite often the conditions in individual experiments must be unsuitable. The experiments that nature provides on a large scale are far more convincing."

Symptoms.—The period of incubation is short and may be estimated

at from three days to a week. The onset is sudden in the form of watery diarrhea affecting from 50 to 100 per cent of the adults and a smaller per cent of the young, but as a rule the calves are not visibly affected. On the first day it may attack only one or two cows. The appetite and milk secretions are greatly diminished and the animals suddenly become gaunt and dull. The temperature is from normal to 103° F. and the pulse is from 65 to 70. In the average case the pulse, respiration and temperature are normal. The feces are watery, fetid, and profuse. The color is usually brown. In some instances the feces have been black and the disease has been termed "black scours." The course is from three days to a week and scouring usually ceases at about the end of the third day. In many herds the disease is so mild that it receives little attention unless there is a distinct fall in the milk-flow. It is rarely fatal and there is no record of a fatal case in our clinic.

In severe attacks the feces may be tinged with blood and even contain clots of blood mixed with mucus. Recurrent attacks have been reported in heavily fed cattle. In the vibrionic enteritis in calves described by Jones and Little[7] the course was usually chronic.

"Immunity or increased resistance as a result of infection probably does not last very long, yet the disease does not often recur in the same herds during the same year."[4]

The *diagnosis* seldom presents any difficulty. In severe types, which are unusual, the presence of blood in the feces may lead one to suspect coccidiosis. Coccidiosis may be recognized by finding many oocysts in the feces, but these are not always present in the feces in the beginning of an attack. A microscopic examination of the feces for vibrios may establish the diagnosis, but this examination requires special knowledge of technic not readily available. In winter dysentery the seasonal incidence and the age of the animals attacked are unlike those of coccidiosis with few exceptions. Severe attacks of coccidiosis in stabled cows usually cause a high mortality, while severe attacks of winter dysentery are rarely fatal. It is probable that many epidemics of winter dysentery in cows have been diagnosed as an "intestinal form of hemorrhagic septicemia," and it is possible that some of the epidemics of unknown cause in older calves are instances of vibrionic enteritis. Experimental transmission by means of a drench containing feces from the sick may identify winter dysentery. In one instance a cow came down with diarrhea shortly after receiving such a drench, and her suckling calf contracted the disease from the dam, but such transmission experiments are often negative. Because of its sudden development, often after the use of a new supply of grain, there is a tendency to attribute winter dysentery to the "feed," but under present methods

of milling, commercial grain mixtures are rarely the cause of disease.

Treatment consists in the administration of creolin 4 to 8 drams (15 to 30 cc.) or other intestinal antiseptic. Copper sulfate (30 to 120 cc. of a 1 per cent solution according to age) is useful. And chlorine (120 cc. of a 4 per cent solution) is also recommended. At the beginning of an attack, when only two or three animals are affected, measures to prevent attacks in the remainder of the herd are desirable. For this purpose the normal animals have been included in the treatment. A weak solution of hydrochloric or sulfuric acid in water sprinkled on the hay has been used. Severe forms are handled as gastroenteritis. Other "favorite treatments" are: two #10 capsules of equal parts pine oil and creolin two to three times a day; or sodium bicarbonate 45 parts, catechu 45 parts, zinc phenosulfonate 10 parts, mix, two to three tablespoonfuls twice daily. The treatment of severe cases is like that of gasteroenteritis: antibiotics and sulfathaladine (2 Gm. daily).

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DIARRHEA IN CALVES

(Scours; White Scours; Calf Septicemia; Gastrointestinal Catarrh; Enteritis)

Diarrhea in the young includes all conditions in which there are frequent loose evacuations of the bowels. As described here it includes white scours affecting chiefly calves in the first one to three days of life and marked by prostration and diarrhea, or by prostration alone, and a high mortality. White scours is thought to be a septicemia or toxemia and it is commonly termed infectious, but complete knowledge of its etiology and pathology is lacking. Since it is often difficult to distinguish between the scours of calf septicemia and other forms of diarrhea they are combined for description. In both human and vet-

erinary medicine neither the frequency nor the cause of diarrhea in the young has been fully explained. As reported by the U. S. Department of Agriculture, of every 100 calves born alive in three dairy breeds, 22 die before they are two years old. When disease is prevalent in a large group of calves the type is often multiple; the younger, from birth to 10 days, suffering chiefly from diarrhea, while among the older, 3 weeks to 3 months, pneumonia is also prevalent. Other conditions in calves are coccidiosis, navel-ill, colds, otorrhea, and sudden prostration and death within 24 hours without showing definite symptoms or lesions—calf septicemia.

Etiology.—Diarrhea is exceedingly common during the first week of life and gradually diminishes with age. In large herds either a mild or severe form may affect every newborn calf over a period of weeks, regardless of the feeding and stable hygiene. It is so common on some farms that it constitutes almost a normal condition, especially during the late winter and spring. While white scours is regarded as the most serious form, other milder forms are frequent, and indicate the presence of conditions favorable to a serious outbreak. The causes include weakness at birth, improper feeding, sudden changes in weather, insanitary pens, continuous use of pens, and unknown probable infections which may be contagious. Attacks of diarrhea with deaths from enteritis are common in the summer months in calves housed in filthy pens and exposed to swarms of flies. In infections outside the intestinal tract, such as colds, otitis, navel-ill, and pneumonia, diarrhea is a frequent symptom. In individuals from one to three months of age, fatal enteritis from no apparent cause is occasional; in such cases the feces may resemble those of white scours or be hemorrhagic as in coccidiosis. Diarrhea is also a symptom of gastrointestinal parasitism, coccidiosis, and winter dysentery. Vibrionic enteritis in calves has been described by Jones and Little.[1] Whenever a calf is sick from any cause, diarrhea is apt to be one of the symptoms. It is especially prevalent among calves purchased from various sources and suckled upon cows for the production of veal. In this group there is exposure to various kinds of infection, the housing and care are often poor, and there is little control of the diet. Some of the most persistent attacks of scours occur in large
diseases may reach from 20 to 30 per cent a year.
ards, yet losses from scours, pneumonia, navel-ill, and miscellaneous
herds where all animals are kept under apparently high sanitary stand-

Infection.—Studies on infection have been directed chiefly to early
of the bacteriology of diarrhea in calves, most writers accept the
theory that white scours is caused by infection, and often the circum-
white scours in calves and to diarrhea in infants. While little is known

stantial evidence is convincing. While it is recognized that various influences, such as season, sanitation, and exposure contribute to the cause, great importance is attached to the action of bacteria which have a normal habitat in the lower intestine, and especially *Bact. coli*. This view is supported by the reports of Jensen[2] (Denmark), and the work of Smith and Orcutt[3] in 1925. The latter observed "a great increase in the lowest third of the small intestine with a spreading of the infection towards the duodenum as the disease gains headway: under these conditions a general intoxication results." Holt[4] refers to this view as applied to infants, and mentions the work of Plantenga and others, who have reported that filtrates of colon bacilli produce toxic symptoms when given by mouth, although without effect when given by rectum; such toxins have been regarded as irritants responsible for the diarrhea. The presence of toxins in bouillon filtrates of *Bact. coli* has also been reported by Smith and Little.[5] The colon bacillus is a facultative saprophyte having universal distribution. Such bacteria are in the stable, in the cow, and in the first swallow of milk. Newborn calves that fail to receive colostrum, that are overfed, or whose vitality is otherwise reduced are especially susceptible. According to this view the colon bacillus is harmless under ordinary conditions, but it may become pathogenic, even causing death quickly in older calves. In recent years doubt has been expressed concerning the influence of the colon bacillus, as described by Smith and Orcutt.[3] But this skepticism is largely speculative, since there is lack of evidence in support of other conceptions. Whether infection in calf diarrhea is intrauterine or occurs after birth, has been a controversial question. But in a herd where diarrhea of the newborn promptly stops as soon as the dry cows are removed to a pasture, the theory of intrauterine infection is difficult to accept.

With advancing knowledge of *viruses* as a cause of disease, it has been suspected that a virus may also be a cause of diarrhea in calves, especially calf septicemia. This suggestion is found in a report by Light and Hodes,[6] who caused diarrhea in calves by infecting them with a filterable agent recovered from infants during an epidemic of diarrhea in hospitals in Baltimore and Washington. By various methods the disease was transmitted from stools, not only from infants to calves, but also from one calf to another. Of 84 calves infected the mortality was about 13 per cent. In calves affected with diarrhea they failed to recover the filterable agent from individuals that had not been exposed to the infant type of infection. In 1943 Baker[7] procured from calves affected with enteritis and pneumonia, by passage through mice, a virus that produced the "characteristic disease" when taken back to

calves. In general, however, efforts to establish proof of a virus as a cause of diarrhea in calves have been negative.

Vitamin A Deficiency.—Observations on the significance of vitamin A have been made by Stewart and McCallum,[8] who report that deaths from *Bact. coli* infection are relatively high in calves of cows whose colostrum has a lower vitamin A content than 250 Blue Units (Moore's) per 100 cc., and that cows' colostrum as distinct from milk, is high in vitamin A. They also observed that in cows on a low vitamin-A diet there was no increase in vitamin A in the colostrum after daily feeding through the winter of either 3 pounds of carrots, or 1/7 pint of cod liver oil (70,000 I.U.). Hart[9] writes of vitamin A deficiency, "In all animals depletion of body stores in pregnant females results in death of fetuses in utero or the birth of weak offspring which frequently die soon after birth. This may be the only symptom of the condition manifested in range cattle. The air passages may become involved, resulting in lung abscesses or a chronic form of pneumonia. Diarrhea is not uncommon and in newborn calves may be confused with white scours."

The theory that lack of certain vitamins is the cause of scours in calves has been reported by Phillips et al.[10] of Wisconsin. Blood plasma of newborn calves was found to be normally low in vitamin A at birth (0.05 microgram per cc.) with a return to normal (0.14) the first day. They reported immediate and dramatic effects in the treatment of scours from the addition of the B complex in the presence of adequate vitamin A, with indications that nicotinic acid was the effective member. In treated animals the feces returned to normal consistency in from 12 to 24 hours and the mortality was reduced to zero. They reported, however, that nicotinic acid and vitamin A did not control diarrhea and septicemia in calves which came down and died within 24 to 48 hours; thus no claim was made for either prophylactic or curative action in early white scours of the newborn. In one herd where calf diseases were persistent I prescribed cod liver oil or shark liver oil and vitamin B complex over a period of three months to calves during the first seven days of life. The results were disappointing. Of 54 calves which received prophylactic treatment, two-thirds suffered from such affections as scours, navel-ill, pneumonia, colds, and purulent otitis, and 29.6 per cent died. Of 15 diagnosed as scours there were 6 deaths and autopsies on 3 were negative; the age was chiefly in the first week from the second to the seventh day. Only one died of calf septicemia within the first 24 hours. Of 9 diagnosed as pneumonia there were 7 deaths at an age of about 2 months each. One-third of the 54 remained normal. In this group there was no indication that the disease was prevented by feeding vitamins A and D and vitamin B complex.

In a report by Norton and associates,[11] "The results of the tests reported show that over a three-year period feeding extra vitamins to newborn calves did not prevent scours nor reduce the incidence, duration, or severity of scours." Spielman et al.[12] have shown that a normal prepartum diet supplemented with one million I.U. of carotene, or one million I.U. of vitamin A daily during the last 60 days of pregnancy may increase markedly the vitamin A and carotene reserves of the newborn calf. Similar reports have been made by Wise and associates.[13]

The value of preparturient administration of vitamin A to cows where mortality in calves from "three-day scours" was high has been reported by Bruce.[14] He administered 500,000 units to cows twice weekly three weeks before calving, and this completely prevented the diarrhea in the newborn. Several unpublished reports on the value of this method have been made from other veterinarians in Massachusetts.

Colostral Milk.—One of the first instincts of a healthy newborn calf is to take colostral milk. Smith and Little[15] have shown that the calf receives protective antibodies in colostral milk, but does not receive them before birth. It has also been shown that the blood of the calf at birth is low in vitamin A, that colostrum is high in this vitamin, and that when a calf receives colostrum shortly after birth its blood contains a normal amount within 24 hours. It is important, therefore, that the calf receive this milk as early as possible after birth. The milk of the dam should be fed for at least three days. A change from the milk of the dam to the mixed milk of the herd earlier than the third to the fifth day may result in an attack of diarrhea. Calves of the Guernsey and Jersey breeds are especially susceptible to disease when deprived of colostrum while Holstein calves may sometimes thrive without it.

The Diet.—Earlier writers attached importance to the feed and feeding as a cause of disease of the newborn, but in general faulty diet has been chiefly regarded as a cause of digestive trouble in older calves. A careful study of the dietetic influence in an epidemic of scours shows that consideration for this alone may check the disease when other efforts fail. A history of an epidemic often reveals that the first calf to die was exposed to faulty feeding. It may be difficult to convince an owner that the natural method of raising a calf on a cow can possibly be wrong, and under normal conditions it may not be. But the high-producing dairy cow and her progeny are an artificial product, and when they are part of a large establishment their environment is far from natural. Overloading with milk during the first twenty-four to forty-eight hours may induce an indigestion in which the colon bacillus or other similar organisms rapidly overwhelm the calf; this may be

fatal in ten to twelve hours from apparent toxemia, or it may lead to typical white scours. It is in the first three days that special precautions are required. The term diet as used here does not refer to the usual care with respect to the quality and quantity of food, but to an exact and systematic method of feeding.

Ingestion of Foreign Substances.—Calves have various habits and tastes. Occasionally a calf at birth has a ravenous appetite. In addition to what the dam supplies it eats bedding or similar foreign substance. It is a common habit and one of which many seem to be unaware. Such individuals are vigorous at birth and the perversion is not explained by any known morbid condition. But it soon leads to one from which recovery is often impossible or incomplete. Unfortunately the damage is not limited to the guilty individual, for, regardless of the origin, scours among calves is contagious and once a calf begins to scour its associates follow suit. In older calves diarrhea may be due to a perverted appetite as shown by preference for filthy bedding instead of hay, or they may even suck the navel of another calf in an attempt to obtain urine; such calves are always unthrifty.

Temperature.—Modern stable practice tends to drive everything except the cow from the stable. This forces the calf to a separate barn where it may be exposed to drafts from open doors or cold walls of concrete or stone, such exposure is a frequent cause of pneumonia and diarrhea.

Intra-uterine Infection.—Calves may be weak or sick at the time of birth, and occasionally one may be born with an inflammation of the umbilicus. In herds where the birth of such calves is frequent, losses among the newborn are heavy, for the raising of these calves is difficult. This condition appears to be most frequent where sterility and breeding diseases are prevalent. The dams of such calves are liable to have retained placentae and septic metritis. And the calves themselves are often covered at birth with yellow feces arising from fetal diarrhea. It has been suggested that nutritional deficiency may be a cause of weakness in calves from cows that are stabled throughout the year without any run at pasture. The progeny from an individual dam may nearly all die of navel-ill, a frequent observation in mares. While there is less evidence of intra-uterine infection or disease in cows than in mares, one occasionally encounters a herd where this seems to be the chief source of a heavy mortality in the young. But even when the cause of the disease is apparently associated with some influence located in the dam, there may be no apparent affection of the reproductive system. At the height of a fatal epidemic of calf septicemia, for example, removal of the pregnant cows to quarters not previously occupied by stock may have no influence upon the mortality. Under such circumstances, one

suspects that the cause resides in the dam, and that possibly some essential factor is missing from the colostral milk.

Pathology.—There are few published reports on the pathology of calf scours, but to draw from human sources one would expect to find fatty liver and tubular damage to the kidneys. As described by Holt,[4] observations on infants show that in diarrhea chemical pathology is of chief importance. Emphasis is placed on inadequate digestion, impaired assimilation by the intestinal epithelium, and rapid peristalsis. Dehydration results from an excessive loss of water and of the minerals Na and Cl. The loss of sodium exceeds that of chlorine, and unless there is a compensatory loss of chlorine through the kidneys acidosis results. But often the urinary function is seriously impaired and little urine is excreted. Dehydration is not a simple water deficiency; it is a deficiency of water and minerals of which the extracellular fluids are composed; it is defined by Holt[4] as a reduction of the interstitial fluids of the body, and is usually accompanied by a reduction of the volume of the blood plasma. Data by Spielman[16] and associates indicate that in diarrhea there is a marked reduction in the absorption of carotene and vitamin A from the intestinal tract.

Often the autopsy is negative. Yet over an extensive accumulation of material, one may find all degrees of acute enteritis from a diffuse hyperemia to an inflammation so intense that the peritoneal surface of the bowel is involved.

In animals that have died after a brief course of one or two days, the dominant lesions are those of a septicemia or toxemia with localization in the digestive tract. The cadaver is emaciated. It emanates a fetid odor characteristic of the disease. The hind parts are usually smeared with feces. The body cavities contain reddish serum. The *serosa* (peritoneum, pericardium, pleura) may be normal, congested, or sprinkled with hemorrhages. The *spleen, liver,* and *kidneys* are often degenerated and the spleen may be covered with ecchymoses. In the digestive system the mucosa of the abomasum and small intestines presents various degrees of petechiae and ecchymoses on a wrinkled and dull surface; one may even find free hemorrhage into the lumen of the bowel. In some there is a marked exudation and a swelling of the mucosa as if the animal had ingested a caustic drug. The intestinal contents may be fetid and gaseous. Occasionally the bowels are light-colored and distended with gas. According to Smith,[5] white scours is essentially an enteritis of the lower intestine, yet in different outbreaks the lesions may vary widely both in location and degree. The mucous membrane of the rectum is usually hemorrhagic and raised into longitudinal folds. When the course is prolonged a week to ten days there may be pyemic changes, such as omphalitis, suppurative pleuritis or

peritonitis, arthritis, and catarrhal pneumonia. It is not rare to find foreign substances such as hair, shavings, or straw in the stomachs.

Symptoms.—In *white scours* the calf is usually normal at birth, and when 48 to 72 hours old it suddenly develops a fetid watery diarrhea; occasionally it is weak and scouring when born, and such individuals usually die. In rare instances it is prostrate at birth, unable to rise, dies within two or three hours without taking colostrum or showing diarrhea, and may present on autopsy a diffuse enteritis. Sometimes the onset is gradual, dullness and anorexia being the first symptoms. Blood-flecked meconium and hemorrhagic spots on the conjunctival sclera are premonitory signs that may precede any other symptoms. In severe attacks there are weakness, dehydration, sunken eyes, rough hair, drooped ears, cool extremities and a medium fever or a subnormal temperature. There may be a discharge from the eyes and nose, giving the appearance of a beginning cold. In some instances sudden prostration leading to death in a few hours, without diarrhea, has been observed; the calf may be found dead in the morning with no evidence of previous sickness. Stiffness when walking is common. The hind parts are usually smeared with fetid material that removes the hair and sets up an inflammation of the skin. If the extremities are cool and the skin clammy a normal temperature has no meaning. The appetite is poor or absent. The abdomen is gaunt and the bowel evacuations are light colored, watery, and gaseous; they may be whitish and pasty and infrequently no diarrhea is present. The usual course is from twenty-four hours to two or three days, with an increasing prostration that ends in death. In certain years the death rate is as high as 90 per cent. Under prompt treatment many individuals recover. Navel-ill is often associated with diarrhea. While navel-ill is often attributed to prenatal infection, a thorough and prompt disinfection of the navel at birth may materially reduce the number of sick calves.

In older calves diarrhea may be the first and only symptom. Often an outbreak of diarrhea in older calves is regarded lightly because recovery is the rule, but after a few days several deaths may occur and for this reason any diarrhea should be taken seriously. In severe types the feces may be green in color due to admixture with biliverdin, and the appearance of greenish feces forecasts a serious disturbance. In some cases and in certain outbreaks, in calves 4 to 6 months or more of age, the feces are black—"black scours"; the course may be as long as a month and terminate fatally in gastroenteritis or exhaustion. This form may be observed in association with pneumonia and bowel catarrh in one animal and bowel catarrh only in another. In one 6-month-old calf the entire contents of the digestive tract were black, and the forestomachs contained gallons of black fluid apparently held

back by stenosis of the pylorus. The mucous membrane of the anterior part of the duodenum was thickened and wrinkled to resemble closely the appearance of Johne's disease, while the rest of the digestive tract was apparently normal. The other occupant of the pen was of the same age and was affected with "black scours" for the same period, one month. On autopsy this animal proved to have extensive pneumonia, but there was no obvious enteritis. These cases illustrate the widely variable postmortem changes in diarrhea in older calves. In another epidemic of "black scours" of unknown cause in a group of 6-month-old calves there was a high mortality after a course of from three to five days; the autopsies showed severe hemorrhagic enteritis in the small intestines.

Injury from ingestion of straw or shavings takes place at an early age, one to three or four weeks, but the effect is lasting in the form of poor condition, stunted growth, long hair, and diarrhea alternating with constipation even on a restricted milk diet. Recovery may be gradual under a milk diet, but the calf is usually stunted. Autopsy reveals the mass of coarse material in the stomachs—reticulum, omasum, abomasum.

Diarrhea may precede calf pneumonia at the usual age of from one to two months. The attack is sudden with a high temperature and depression, a syndrome that may bring from the attendant a diagnosis of "intestinal grippe." Within one or two days distinctive symptoms of pneumonia appear, and it is probable that pneumonia is the primary affection. A fatal diarrhea in 6- to 8-month-old calves shipped for a long distance and changed abruptly to a diet of dry roughage, led to a fatal diarrhea with no obvious changes on autopsy. Infrequently older calves, up to 3 or 4 months, under excellent standards of sanitation, may be taken suddenly with a severe diarrhea which resembles in all respects that of white scours of the newborn and terminates fatally in three or four days. In these, also, the postmortem may be negative or pneumonia may be present.

In this miscellaneous group of affections, *diagnosis* according to the bacterial cause is impossible since the nature of the infection is unknown. One may possibly determine whether the cause is faulty diet or housing, or infection, or overcrowding, but even this may be difficult. There is, however, a definite and important distinction to be made between mild and severe diarrhea at any age. It is also important to distinguish between an individual suffering only from diarrhea, and one in which diarrhea is a symptom of some other disease, chiefly pneumonia. In most cases this is not difficult; pneumonia usually occurs in older calves, 3 weeks and over, while primary diarrhea is most prevalent within the first week.

Prophylaxis.—Experience has shown that prevention may be accomplished by means of a systematic method of feeding in combination with the use of muzzles to control the ingestion of foreign material. Briefly it is as follows for a calf apparently normal and vigorous at birth:

1. The cow should freshen in a clean parturition stall. Such stalls are valuable in the control of all acute infections incident to parturition. On large breeding farms, and in purebred herds they are an economic necessity. But regardless of the circumstances of birth the offspring should receive colostral milk at the earliest possible moment. In our practice it has been observed that if a normal calf remains with the dam for twelve hours it obtains sufficient colostrum and does not obtain too much milk. The modern dairy cow gives more milk than is needed by any single calf. When a newborn calf with a ravenous appetite becomes gorged with milk, all the circumstances for the development of a colon septicemia are favorable—the stomach is overloaded, the intestines contain meconium, and the digestive tract is not yet able to function actively.

Some authors attach importance to washing and disinfection of the external genitals and douching the vagina previous to birth, and to receiving the newborn calf on a sterile sheet. These precautions are difficult to apply, and are entirely unnecessary. It is highly desirable, however, that an experienced attendant be present at the time of birth to exert traction when the presentation is normal and the delivery slow because of an oversized fetus, to prevent the cow from lying with the rear end against the wall during expulsive efforts, to disinfect the navel immediately after birth, and, when necessary, to assist the newborn calf in obtaining the colostral milk shortly after birth. Prompt assistance is often necessary to secure a live calf from a posterior presentation. Failure to provide such assistance may result either in death at delivery or a weakened calf that succumbs within the first week.

Disinfect the navel by placing it in a 2-ounce wide-mouthed bottle half filled with tincture of iodine. This is best done with the calf in a recumbent position; the bottle can then be inverted over the region of the navel and pressed against the abdomen until the navel and the surrounding skin are thoroughly saturated. Do not use the same iodine more than once.

2. At the end of twelve hours the calf is muzzled and all food is withheld for the next twenty-four hours. At the end of the fasting period the meconium has passed, the stomach and bowels are relatively empty, and the animal has not suffered any discomfort. When the fasting period ends at night a quart of milk and a pint of lime water warmed to body

temperature are fed. Muzzle sizes for Guernsey calves are: top diameter, 4.75 inches; bottom diameter, 3.50 inches; height, 4.25 inches. Muzzle sizes for Holstein calves are: top diameter, 5.25 inches; bottom diameter, 4 inches; height, 4.50 inches.

3. The regular feeding day begins on the morning of the third day, thirty-six hours after birth. Thus a calf born in the morning would remain with the dam until night and be fasted until the following night, when it would be fed a quart of milk and a pint of lime water. A calf

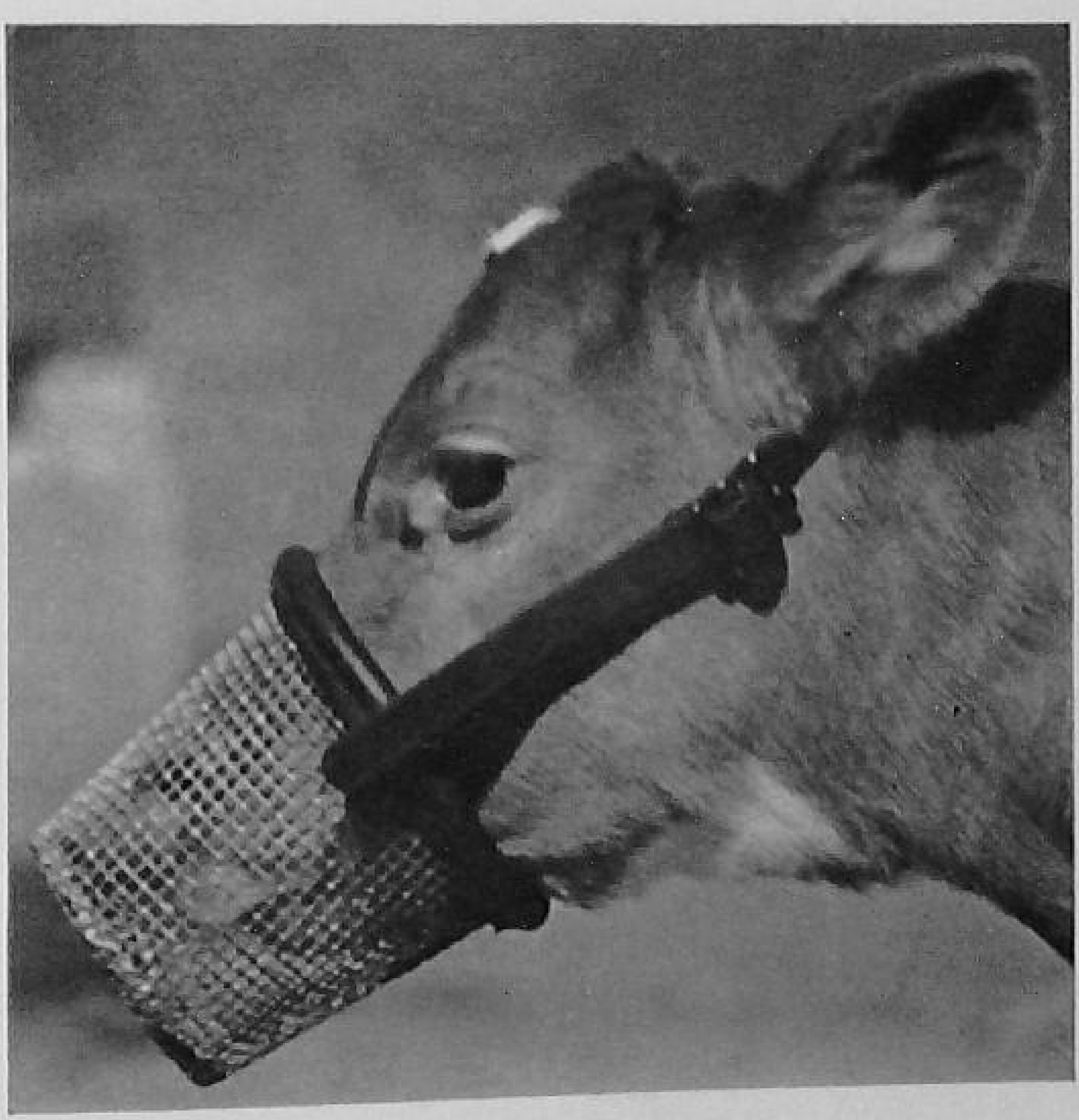

Fig. 18.—Calf muzzle.

born in the evening would remain with the dam during the night and be fasted from the morning of the second to the morning of the third day. The amount of milk allowed on the third day, the first regular feeding day, is about 6 per cent of the weight of the calf, using the dam's milk. For the Guernsey breed, with a birth weight of 65-70 pounds, this is 4 pounds. It is preferably divided into three feedings, to each of which is added a pint of lime water, but it is not usually practical to feed three times a day. The amount of lime water added to each feed is never more than one pint. A daily increase of 8 ounces of milk for the first three weeks will constitute a suitable addition. At the end of the first week the calf may receive 8 to 12 per cent of its body weight of

milk daily. During the first week, take the temperature before the noon feed. If it is 103° F. or more give an enema and three ounces of liquid petrolatum and withhold food until the temperature is normal and the calf ravenously hungry.

Weak and undersized calves need to be fed often and kept on a decidedly restricted diet until they become strong and vigorous. The early fasting period should be omitted. It is helpful to supplement the low milk diet with olive oil (4 ounces daily). If the newborn calf is unable to rise and suckle, it may receive 8 ounces of the dam's milk three to five times daily. Prompt administration of the dam's blood (50 to 100 cc. subcutaneously) sometimes results in marked improvement in these cases. It should be given as soon as possible after birth. In herds where the calves are commonly born weak or sick the mortality is high under any circumstances, and preventive measures involve a survey of the breeding and nutritional condition of the entire herd.

Some dairy farmers raise calves on nurse cows, but this method has not proved generally successful, since the calf may overeat and still fail to remove sufficient milk for the health of the udder. These objections may be overcome by assigning one calf for each 10 pounds of milk produced daily by the cow, and under these conditions a diseased udder may improve. Calves that become unthrifty and develop scours after eating coarse indigestible roughage may improve rapidly when placed on a nurse cow.

Housing should protect calves against extreme cold, sudden changes in temperature, and cold drafts from open doors. They should be kept dry, warm, and clean. If the calf barn is large and the control of temperature is imperfect, small blankets made of heavy sacking may be used; these are either sewed or tied to fit the body closely and are worn for about two weeks. A suitable temperature in calf barns is 45 to 55° F. Individual pens may be desirable for the first few days, but small tightly boarded pens are difficult to ventilate and they predispose to unthriftiness and pneumonia after the first two weeks. Where muzzles are used, the dangers from mingling are greatly reduced, and calves thrive when kept muzzled and on a milk diet for the first thirty days.

Losses from white scours may be reduced by allowing the cows to freshen at pasture or in open fields, and excluding the calf from the barn for at least ten days. Newborn calves may be protected against fatal epidemics by removal of the cows before freshening to quarters not previously occupied by live stock, or even to a neighbor's stable, but in such cases persons who have worked on the infected premises must not enter the new quarters.

CALF FEEDING RECORD

Directions.—Disinfect the naval at birth. Leave the newborn calf with its dam for twelve hours and see that it receives colostral milk shortly after birth. At the end of twelve hours, apply a muzzle and fast the calf for twenty-four hours, if it is not weak. At the end of twenty-four hours, begin the regular feeding ration, feeding twice daily (pints or pounds) as follows:

DATE	FEED DAYS	SWEET MILK	LIME WATER	SKIM MILK	NOTE EVERYTHING ABNORMAL
	1	2	1		DAM'S MILK
	2	2	1		DAM'S MILK
	3	2	1		DAM'S MILK
	4	3	1		DAM'S MILK
	5	3	1		DAM'S MILK
	6	3	1		
	7	3	1		
	8	3	1		
	9	3	1		
	10	3	1		
	11	4	1		
	12	4	1		
	13	4	1		
	14	4	1		
	15	4	1		
	16	4	1		
	17	4	1		
	18	4	1		
	19	4	1		
	20	4	1		
	21	5	1		
	22	5	1		
	23	5	1		
	24	5	1		
	25	5	1		
	26	5	1		
	27	5	1		
	28	5	1		
	29	5	1		
	30	5	1		BEGIN TO FEED HAY AND GRAIN
	31	5	1		
	32	4	1	1	
	33	4	1	1	
	34	3	1	2	
	35	3	1	2	
	36	2	1	3	
	37	2	1	3	
	38	1	1	4	
	39	1	1	4	
	40		1	5	

It is obvious that not all of these directions are suitable for average conditions, but where losses among valuable animals are heavy, extreme measures may be necesssary. The schedule on this page has proved to be helpful where many calves are being raised. A record of each animal is kept. The amounts are adjusted for the Guernsey breed. Records of this kind will demonstrate whether or not there is any relation between unthriftiness in calves, and sterility and abortion in such individuals at maturity.

Open Pens.—After the usual methods of prevention have been observed, diarrhea may still persist in combination with a miscellaneous group of other affections. Under such circumstances the cause may be found to reside in some obscure fault in housing or nutrition. In regard to housing the risk of continuous operation of maternity stalls and calf barns, especially in large units, has long been recognized. And there is evidence that continuous operation of small units or calf pens may result in an accumulation of obscure unfavorable conditions which may not be controlled by cleanliness and frequent disinfection. This is a frequent observation in human maternity hospitals where the precise cause may also be elusive. On a number of farms marked improvement has followed the housing of newborn calves in pens or sheds open on one side, preferably the south, combined with feeding on nurse cows. The calves are rationed at the rate of 10 pounds of milk per day for Guernseys or Jerseys; thus a cow producing 20 pounds would nurse two calves. In order to obtain the essential colostrum the calf is allowed to suckle its dam for three days, when it is removed to a well bedded nurse-cow pen, even in zero weather, where it remains until 4 months old. While this method does not prevent white scours which commonly appears on the second or third day, calves raised under this system are more vigorous and apparently more resistant to disease than pail-fed animals, and they are remarkably free from respiratory affections. After weaning the habit of sucking other calves is rarely observed. Cows that do not qualify for the milking line because of appearance, lameness, defective udders, etc., may be used as nurse cows. While there may be objection to the use of milk from cows with diseased udders, experimental calves raised on mastitis milk have been free from mastitis at the time of calving.

In conclusion, one seems justified in the opinion that a condition so prevalent as diarrhea in calves is due to a wide variety of causes, and that the calf itself harbors the elements, either in the form of infection or nutritional weakness that make for a narrow margin between health and disease. Belief that the gastrointestinal tract is an important portal of entry of infection is supported by the remarkable curative

and prophylactic effect of sulfathalidine, whose action is limited to the digestive system, and the fact that Light and Hodes[6] induced diarrhea in a calf by introduction of the infant strain of virus through a stomach tube. The view that diarrhea in calf pneumonia is not secondary to the pneumonia, is supported by the observation that when pneumonia has been brought under control, as in open nurse-cow sheds, diarrhea may continue to be widely prevalent without respiratory combinations.

Sulfonamides.—Since the introduction of sulfonamides that have a bacteriostatic action upon intestinal bacteria and are not toxic, such

Fig. 19.—Open pen method of raising calves on nurse cows.

drugs have been administered to newborn calves as a means of preventing white scours. According to Thorp[17] both sulfathalidine and sulfasuxidine had more effect than sulfaguanidine on coliform organisms in the gastrointestinal tract of calves, and effective dosage of sulfathalidine was about one-fourth that of sulfasuxidine. Furthermore, there have been no reports from toxic action of sulfathalidine when given in large amounts. This is important in the use of an agent that may depend on widespread distribution in the intestine to overcome or prevent massive infection. Sulfathalidine has proved to be highly useful as a prophylactic against white scours and it may either prevent the attack or limit its severity. Wise and Anderson report that following a mortality of 71.4 per cent of all infected animals, 89.5 per cent of 38 calves

were protected. They advise 4 grams daily in two equal doses over three or four days beginning between 6 and 12 hours after birth, and suggest an increase in dosage and frequency in severe outbreaks. Udall[18] used 8 Gm. daily for the first two days, followed by 4 Gm. daily on the third and fourth days, in two divided doses, a total of 24 Gm. The first dose is given as early as convenient after birth; in the period under observation there has been a distinct decrease in the number of attacks of early scours. It is possible that even one 4-gram bolet at birth may be an adequate prophylactic dosage. Sulfathalidine is conveniently administered in 4-gram bolets or ¼-ounce capsules with a ½-inch balling gun.

Antibiotics.—Among the antibiotics which may act as preventives against diarrhea and calf septicemia in calves are streptomycin (dissolve 1 Gm. in 8 cc. of water, give one half orally and one half subcutaneously); soluble penicillin 2 million units, plus streptomycin 2 Gm., subcutaneously; terramycin, one capsule orally containing 100 mg. as soon as possible after birth; aureomycin, 5 mg. per lb. body weight per vein (reported to be a specific in otorrhea in calves). Except in herds where calf septicemia is active and affecting nearly every newborn calf, the routine use of either sulfonamides or antibiotics is not indicated.

Treatment.—Sulfathalidine: In white scours (calf septicemia) one has to deal with dehydration, enteritis and intoxication (toxemia). In mild forms there may be symptoms of diarrhea only, with recovery after a few hours, but in severe attacks only vigorous treatment at the onset is effective. On the theory that enteric infection with coliform organisms is the cause, emphasis has been placed upon the use of so-called intestinal antiseptics, and more recently on the bacteriostatic action of sulfathalidine. In the selection of sulfonamides, precaution should be taken to avoid damage to the kidneys which may already be impaired from the diarrhea. The effect of either diarrhea or sulfonamide intoxication is to suppress the normal functions of the kidneys, and consequently a "therapeutic" dose of a sulfa compound capable of entering the circulation may do harm. The "therapeutic" dose of such sulfonamides has been estimated at about 1 grain per pound of body weight—6.5 grams per 100 pounds. One cannot foretell, however, what fraction may be deposited in the form of crystals in the kidneys, where it may cause crystalluria, hematuria, and uremia. Since sulfonamides capable of entering the circulation have become household remedies it is probable that unrecognized cases of poisoning occur. In the treatment of diarrhea it is desirable that the action of the sulfa preparation be limited to the alimentary tract, that it possess active

bacteriostatic action, and a wide range of dosage. All of these requirements are met by sulfathalidine. While a therapeutic dose of from 6 to 12 grams per 100 pounds of body weight for a period of from 3 to 5 days has been generally recommended, observations on the treatment of many cases indicate that in severe attacks increasing dosage yields corresponding benefit. In an attack without general symptoms a minimum dose brings prompt results; there is improvement within one or two days. Higher efficiency may be attained when the routine dosage is set at 30 grams daily in two divided doses. In severe and sudden attacks prompt and massive dosage may be required; thus very sick animals less than one week of age have recovered under daily dosage of 60 grams given in four divided doses. Where this drug has been used freely there has been a marked reduction in the mortality. Such striking and prompt results from an agent that acts entirely within the alimentary tract suggests that diarrhea in calves is an enteral infection as reported by Jensen[2] in 1892 and by Smith and Orcutt[3] in 1925.

Antibiotics, as recommended under prophylaxis, are apparently equally effective in the treatment of calf septicemia. These include especially those which are active against gram-negative infections: streptomycin, terramycin, aureomycin, and chloromycetin (500 mg. orally 2-3 times daily).

In early scours with dehydration and prostration one needs to apply the principles followed in the treatment of infants in order to supply the loss of fluids and minerals and restore the action of the kidneys. For this purpose one may administer normal saline solution (500 cc.); or dextrose (200 to 500 cc. of a 40 per cent solution); or blood transfusion; these may be given per vein or subcutaneously, or both. The subcutaneous injection of 100 to 150 cc. of antihemorrhagic septicemia serum, followed by 50 cc. daily, has been followed by recovery from severe attacks of scours when combined with large doses of sulfathalidine. In these cases it is probable that benefit resulted from the general action of the blood plasma and not from the presence of specific antibodies.

Calf scour serum (antibacterial serum—bovine) has been widely used both as a prophylactic and a cure. Often it confers no apparent benefit. I have not observed that it possesses either prophylactic or curative action. The dam's blood is more effective in doses of 250 to 500 cc., defibrinated by drawing 500 cc. of blood into a bottle containing 1.5 to 2 Gm. of sodium citrate; administer intravenously or subcutaneously. Or one may transfer the blood direct from dam to calf.

In older calves the diet should be reduced to about half the usual amount or withheld entirely. Evacuation of the bowels is indicated

when they are overloaded with milk or contain fermenting material. Olive oil 2 to 4 ounces (60-120 cc.) combined with bismuth subnitrate (20 Gm.) three times daily is usually effective. In older calves, 2 to 4 months, castor oil 2 to 4 ounces (60-120 cc.) may be indicated when there is evidence of decomposing intestinal contents as shown by fetid fluid feces and possibly bloat. In delicate young calves olive oil or mineral oil is superior to castor oil. After the bowels are rid of an excessive load of irritating material, attention should be given to the sensitive mucosa. For this purpose bismuth subnitrate (Gm. 10 to 20 twice daily) is excellent. Stimulants and carminatives are indicated from the beginning: aromatic spirits of ammonia 4 drams (16 cc.) in 12 ounces (360 cc.) of soda water every four hours; soda water consists of 1 ounce (30 Gm.) bicarbonate of soda to 1 pint (500 cc.) of water. Other useful stimulants are strychnine sulfate 1/30 grain (0.0022 Gm.) twice daily, or camphorated oil 5 to 20 cc. once or twice daily. The following, or a similar mixture, is often used in simple diarrhea in calves:

Tincture of opium, camphorated	6 minims	(0.370 cc.)
Pepsin	4 grains	(0.259 Gm.)
Salol	2 grains	(0.130 Gm.)
Bismuth salicylate	8 grains	(0.518 Gm.)
Spirits wintergreen	10 minims	(0.616 cc.)
Water q.s.	1 ounce	(30.000 cc.)

Return to the normal diet needs to be gradual. It is important to consider the nutritional requirements, but there is danger of a relapse from an overfeed. The minimum amount of nourishment is represented in barley or linseed meal water, made by pouring 3 parts of boiling water over 1 part of the ground grain. Stir, cool, and give 8 to 16 ounces every 3 to 4 hours. Usually the calf is able to take a half pint of heated or boiled milk three to four times a day at intervals of three to four hours. If the animal can take this amount, the milk may soon be increased to 4 to 6 per cent of the body weight. Calves that are undersized, or that have lost through sickness until they weigh no more at the end of a month than they did at birth, should be fed according to weight rather than age.

To check mild forms of scours in older calves the following is beneficial:

℞ Spirits ammon. arom	℥ iii (90 cc.)
F. E. Capsici	℥ i. (30 cc.).

M. Sig. Tablespoonful in 12 ounces of soda water every 2 to 4 hours.

Older calves with perverted appetite may be placed in individual pens

with no bedding; if they are not too old a return to milk may be beneficial. In severe "black scours" in older calves there may be failure to improve under any of numerous methods of treatment. Sulfathalidine is often highly effective in the treatment of diarrhea in older calves.

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TRANSMISSIBLE GASTROENTERITIS IN BABY PIGS

Definition.—Transmissible gastroenteritis in baby pigs is an acute rapidly fatal diarrhea in newborn pigs caused by a filterable virus. The specific cause was identified in 1946 by Doyle and Hutchings[1] of Purdue University, Indiana, who reported that for several years they had observed sporadic outbreaks of fatal diarrhea and vomiting in newborn pigs with less frequent diarrhea and recovery in older animals. While they do not regard it as the most important form of baby pig mortality,[2] they believe it is increasing, and this description is compiled from their reports.

Etiology.—On affected farms the presence of the disease is irregular. As in "baby pig" disease in general, and in affections of the newborn in other species, the malady is intensified by the use of a large central maternity unit. Thus in one instance only 90 of 390 pigs survived, the age at death ranging from two days to a week. Apparently it spreads from baby pigs and sows to shoats, yet it may be found in shoats on farms where there are no baby pigs. In experimental transmission it was observed that the disease developed promptly in baby pigs following contact exposure in pens or by putting portions of the gastrointestinal tract in the mouths of healthy pigs. The onset appeared within 24 hours after exposure with a mortality of 95 per cent within five days. After feeding 0.5 per cent of filtrate the mortality was 50 per cent. Bacteria-free filtrates from the gastrointestinal tract and other tissues of infected pigs were infective in pigs from 1 to 9 days old when introduced through various channels in dilutions as high as 1:1000. Hog cholera serum afforded no protection. In one severe outbreak purchased sows from a local sales barn were thought to be the cause.

Pathology.—With few exceptions gastritis and enteritis with engorgement of the mesentery are present. Often the mucous membrane presents a deep cherry red color that is visible through the peritoneal surface. The kidneys are degenerated. The most constant finding is an abundant intestinal fluid which may be whitish, yellowish, or greenish in color.

Symptoms.—"During the past several years we have observed sporadic outbreaks of the disease characterized by vomiting, diarrhea, dehydration, rapid loss of weight and high death loss in 2-7 days in young pigs. In older swine, diarrhea, vomiting, and loss of appetite are the most characteristic symptoms. The most consistent symptom in older swine is diarrhea. Recovery in older swine is rather prompt and the mortality is low."[2]

"The blood cell studies of affected pigs showed 49 per cent increase

in leucocytes on the fourth day after exposure. . . . The neutrophils were increased by 21 per cent and lymphocytes decreased by 28 per cent."[2] The death rate decreases as the animals get older. Sulfamethazin and streptomycin were ineffective.

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GASTROENTERITIS

Definition.—A severe acute inflammation of the stomach and small intestines, less often the colon and cecum, characterized by congestion and hemorrhage of the mucosa as well as by various degrees of toxemia. The symptoms are prostration, frequent shallow breathing, and watery fetid feces rich in mucus or mixed with blood. The term includes both gastritis and enteritis for both are usually present, and often a clinical differentiation is impossible. The condition may be primary or secondary, partial or terminal. The following clinical types may be recognized: (I) Primary dietetic. (II) Infectious. (III) Toxic, as from metallic poisons (lead, arsenic). (IV) Parasitic. (V) Traumatic.

Occasionally a cow which dies suddenly, or that is found dead in the morning, may on autopsy show nothing except enteritis; this may be limited to the colon or involve the entire gastrointestinal tract. As an example, a cow ate normally and gave 40 lbs. of milk at 6:00 P.M., and was found dead three hours later; on autopsy the terminal section of the colon was dark colored for a distance of 20 feet, no other lesion could be found, and the cause was not determined.

Dietetic Gastroenteritis

Etiology.—This is a primary form that may result from indigestion, acute gastrointestinal catarrh, or proceed from the onset as a severe inflammation. The *seasonal occurrence* is that of indigestion; in cows it is exceptional from June to September. It occurs after eating spoiled food, such as grass that has been repeatedly frozen, heated freshly cut grass, corn, or silage, and recently cut heated mouldy silage. In bovines it is the usual fatal termination of neglected indigestion and gastrointestinal catarrh. The *young* of all species, especially calves and pigs, are often attacked. *Predisposing causes* are found in exposure to moisture and filth. The remark that "calves will not thrive unless their feet are kept dry," implies affection of the digestive tract.

A rapidly fatal form occurs in animals that receive insufficient food, water, and rest during shipment,[2] and are then allowed too much food and water on arrival at their destination. Horses and mules that have been fattened rapidly in pens previous to shipment are especially predisposed to gastroenteritis in transit. In horses and mules in transit it is important to observe the regulation that animals shall be unloaded for feeding, watering, and rest at the end of each 28-hour period; that they receive their solid food, hay and rest at least one to two hours before they are watered; and that before they are allowed in the pens, troughs are drained and the remains of previous feedings are removed from the racks. The minimum allowance of 10 pounds of hay per day is not sufficient. Two to three bales of hay per car should be scattered along the wall for the horses to eat in transit. This type has been observed in army horses and attributed to poisoning or anthrax because of the rapidly fatal course and the numbers affected. Bacteriological investigation by Graham[1] of such outbreaks has shown that *Salmonella-enteritidis* obtained from dead animals proved fatal to experimental mules fasted and subjected to conditions inducing fatigue, and that experimental animals not fatigued by fasting were not susceptible to such infection.

In sheep, it has been observed that a sudden change from short pasture to a full diet of alfalfa and grain has been followed by peracute fatal enteritis—see overeating in lambs, p. 124. *Infection* is doubtless active in all dietetic gastroenteritis, but the primary cause is often some irritant in the food. Saprophytic bacteria may cause fermentation in food or intestinal contents, and the chemical products of such fermentation may act as the immediate cause of the inflammation.

Morbid Anatomy.—In enteritis associated with transportation in horses the chief changes are in the digestive tract. The bowels are distended with gas. The contents of the stomach and intestines consist of a varying amount of liquid and practically no food; there are few conditions which leave the digestive tract so completely empty. In most cases there is a diffuse hemorrhagic enteritis affecting the greater part of the mucosa, but in some the hemorrhage is circumscribed. The liver, kidneys and spleen may be normal or degenerated; often the kidneys are badly degenerated. Intermuscular hemorrhage is frequently found in the large skeletal muscles of the neck and gluteal regions.

On postmortem examination of sporadic cases of enteritis in mature animals one finds extensive hemorrhagic inflammation of either the small or large intestines, or both; in some, the stomach is also hemorrhagic.

Symptoms.—The onset is variable, depending somewhat on the age

and the cause. In the mature, dietetic irregularities leading to bowel catarrh and more serious inflammations have a history of one to three days of dullness, anorexia, and green, black, fetid or bloody feces. In severe inflammation, the onset is often painful and sudden, soon leading to rigors and collapse; this type is suggestive of a toxic or infectious origin.

The *attitude* often is recumbent, the animal may rise promptly, but appear weak—an unfavorable sign. Dullness is common to all. Grinding the teeth, delirium, convulsions, and orthotonus are terminal symptoms. In cows, the back may be arched as in acute vaginitis. Moderate or severe pain is constantly present; in bovines it is manifested by cautious stiff movements, reluctance to walk, an expiratory grunt, or carrying the tail slightly to one side. Horses show intermittent colic. The expression is anxious or staring.

The *eye* is often sunken and the conjunctival mucosa pale or icteric, though it may be congested in gastritis. Marked congestion and icterus are more frequent in equines. A serous or mucous discharge from the eyes and nose, as well as swelling of the lids, is an occasional septicemic symptom in cows. Emaciation proceeds rapidly, and finally, in fatal cases, the skin becomes doughy and remains in folds when raised.

Fever, as recorded per rectum, is rarely present, except that horses may have a high temperature at first; it is normal within a few hours and subnormal one or two days before death. There are depression, anorexia except for water, cool extremities, often chills, and fast pulse and breathing. The *pulse* is 75-80 or more. At 24-hour periods the frequency may vary widely, falling from 80 down to 60. When a drop in the pulse is accompanied by other favorable signs it indicates improvement, but taken alone it may be deceptive. This tendency of the pulse rate to slow down in fatal gastroenteritis is also observed in peritonitis. The quality of the pulse (size, strength, hardness) and rhythm are abnormal. The rate is higher in horses than in cattle. A rising pulse combined with a falling temperature is a bad sign; it indicates circulatory failure. The *rate of breathing* in cows is from 20-40, in severe attacks even 60; it is always shallow and serves as a useful prognostic sign. In horses it may be slow, for example, pulse 108, respirations 7, temperature 102.8° F. forty-eight hours before death.

Digestive System.—With few exceptions, anorexia is complete. Partial appetite may remain when the stomach is not affected, while horses often manifest great thirst. In the less rapidly fatal forms food may be nibbled occasionally. The mouth is clammy, cool, often fetid, and froth may collect between the lips. A tense gaunt abdomen is frequent. In cows, the movements of the rumen and intestines are weak, and there

may be complete paralysis. In horses loud gurgling sounds (borborygmi) are often heard. Soreness on percussion or kneading over the affected organs is common. Because of the tense abdominal wall in horses, pain on palpation is usually not induced. The *feces* reveal the nature of the disease and the character of the inflammatory exudate in the majority. In cows, mucus is often abundant; the evacuations are thin to watery, discharged involuntarily, green, black, or blood-tinged, and fetid. In deep inflammation of the colon, clots of blood may appear; in severe inflammation of the small intestines there may be large amounts of gelatinous mucus. A hemorrhagic inflammation of the forestomachs and colon may be associated with absence of defecation, and dark, nearly normal feces may be evacuated when the small intestine is only slightly inflamed. Retarded evacuation is due in part to paralysis of the digestive tract, in part to toxemia. The character of the feces depends on the degree of inflammation, and the quantity upon the degree of paralysis. In gastroenteritis in *calves*, evacuations are fluid or pasty, gray or yellow, fetid, and often mixed with gas. In horses the feces are brown, fetid, semifluid or watery, gaseous, and passed with tenesmus or involuntarily.

In horses and mules that sicken in transit, from one to several may be affected. They may sicken in the car or not until forty-eight hours after unloading. The records show that the animals reach the first feeding station in good condition, but when the trip is continued for from thirty-six to forty-eight hours, and they are again fed, watered, and rested, cases begin to appear. At first there are inappetence, dullness, and congestion or jaundice of the conjunctival sclera. These symptoms are soon followed by weakness, pendulous lips, and colic. The temperature may be normal or as high as 105° F. The pulse and breathing are rapid. The bowel evacuations are copious, watery, and fetid. Tenesmus is marked and may be extreme after the bowels are completely empty. Ulceration of the oral mucosa and laminitis are frequent. The course is from eight to twelve hours.

Course and Prognosis.—In two to four days after the disease is well established the patient is usually better or beyond recovery. The mortality is high, fatal forms lasting about a week; violent attacks may lead to death in three days, while less intense forms last for two weeks. In 4- to 6-months-old calves a subacute enteritis may persist for a month or more.

Diagnosis.—Gastroenteritis is diagnosed by the general symptoms, tenderness over the abdomen in cows, and the character of the feces. *Acute catarrh* is often associated with acute indigestion and merges imperceptibly into deeper inflammations; it is not always possible,

therefore, to make a restricted pathological diagnosis. In cows, gastroenteritis may be associated with *peritonitis*: differentiation is based on consideration of the etiology, the absence of diarrhea in peritonitis, and the pain which is greater in peritonitis. *Metritis* leading to sepsis and diarrhea is suspected by the parturient condition, and may be confirmed by a vaginal examination if the fetus has been expelled. *Infectious diseases* show a higher temperature, affect more individuals, and occur in animals eating wholesome food.

Treatment.—The problem in treating dietetic gastroenteritis is to remove irritating material from the bowel, control fermentation, protect the mucosa, and combat intoxication. Treatment needs to be started early in order to anticipate and prevent complications. *Laxatives* are most advantageous in the beginning; after the bowel has been emptied by purging, their value is questionable. Equal parts of mineral oil and castor oil 1 to 2 pints (500-1000 cc.) repeated according to individual requirements, or mineral oil 1 to 2 quarts (1 to 2 liters) every twelve to twenty-four hours are suitable for large animals. *Fermentation* may be controlled with intestinal antiseptics, of which creolin 4 to 8 drams (15-30 cc.) is excellent, or one may prescribe antacids, such as lime water, bicarbonate of soda, or aromatic spirits of ammonia 4 to 8 drams (15-30 cc.). As a *protective*, bismuth subnitrate 3 to 4 ounces (90-120 Gm.) in 1000 cc. of warm milk is especially recommended in severe irritation in cows. Tannic acid 1 ounce (30 Gm.) daily is widely used as an astringent and protective. Mineral oil 1 to 2 quarts (1-2 liters) daily, also protects the mucosa and mechanically absorbs debris. Whatever fluid is selected may be given through the stomach tube. Sulfathalidine is indicated, and it may be combined with streptomycin (2-6 Gm.) per 100 lb. body weight in two divided doses.

Prevention and control of weakness, due to resorption of toxic products and loss of water, is highly important. Marked weakness and collapse with increased frequency of breathing and pulse and a falling temperature are forerunners of a fatal termination. Keep the patient strictly quiet, and, in cold weather, warmly clothed. Nourish with water of oatmeal, barley, or linseed; this is made by stirring 1 part of the meal with 3 or 4 parts of boiling water and decanting the liquid. Such mucilaginous drinks, one or two quarts at a time at intervals of about three hours, are useful. Weakness is combatted with stimulants, such as camphorated oil 1 to 2 ounces (30-60 cc.) injected into the pectoral muscles; caffeine sodium benzoate 2 to 4 drams (8 to 15 Gm.) subcutaneously; intravenous injections of normal saline solution, or slow prolonged enemas of normal saline; and blood transfusion. Dextrose (500 to 1000 cc. of a 40 per cent solution) intravenously has won

favor in a variety of septic conditions, of which gastroenteritis is an example. Because of its caffeine, black coffee is also useful.

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Infectious Gastroenteritis

Etiology.—Infectious gastroenteritis, and enteritis, occur under the following conditions: (a) As a *partial condition* in anthrax, hemorrhagic septicemia, hog cholera, influenza, swine erysipelas, and other similar acute infections. (b) *Secondary* to septic metritis and mastitis, traumatic gastritis—rarely, intestinal obstructions, parasitism, and toxic conditions in general. (c) *Primary* in white scours in calves—*Escherichia coli*, coccidiosis, infectious enteritis in swine (*Salmonella choleraesuis*—Biester and Murray),[1] winter dysentery and other forms that reveal the cause of *primary infection.* An outbreak of paratyphoid dysentery, involving over 30,000 lambs, with a loss of 2,000, has been described by Newsom and Cross.[2] An organism of the paratyphoid group was isolated, and the disease was reproduced experimentally in normal lambs. Fasting was shown to be an important predisposing cause. In the primary group may also be included severe forms of winter dysentery in cows, and enzootic and sporadic attacks of enteritis that are apparently infectious but yield no causative organism. Enteritis in sheep in the Yellowstone Valley caused by the protozoan parasite *Globidium gilruthi* has been described by Marsh and Tunnicliff.[3] Frequent examples are epidemics of scours in calves from one to six months old. Obscure enteritis may theoretically be attributed to intestinal bacteria that act when the resistance is lowered, or to pathogenic bacteria that enter in the food or water. (d) Exhaustion from forced marches, long distance races, and transportation is sometimes followed by a rapidly fatal hemorrhagic type that closely resembles the acute general infections. Attacks following transportation are usually the result of faulty feeding, watering, and rest.

Symptoms.—These vary according to the nature and virulence of the infection. In most essentials they conform to other types, though a higher and more prolonged temperature is the rule. The onset is usually sudden and characterized by a high initial temperature and a weak irregular pulse; finally the pulse increases while the temperature falls.

The disease may be enzootic, and it bears no relation to the quality of food. In making a diagnosis, consider the possibility of improper food or feeding, the seasonal occurrence of the dietetic form, the seasonal occurrence of specific infections (anthrax, hemorrhagic septicemia), the possibility of poisoning (lead, arsenic, bracken), and of contaminated water.

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Toxic Gastroenteritis

This is an intense form induced by the ingestion of such poisons as corrosive sublimate, lead, arsenic, and nitrate of soda. One may also include the eating of ferns and brakes—bracken poisoning. When a single animal in a herd suffers without apparent cause from gastroenteritis that terminates fatally in one to three days, one should search for poisoning. Painted walls, or an old painted board, a paint bucket thrown into the bushes in the pasture, or a discarded container of arsenate of lead spray are often found. Nitrate of soda is especially violent; it is eaten from the ground after being spread for fertilizer, from sacks left carelessly within reach of stock, or it may be mistaken for salt. Toxic gastroenteritis has followed the administration of vermifuges, such as male fern, copper sulfate solution, carbon tetrachloride, oil of chenopodium, and carbon disulfide.

Symptoms.—Toxic enteritis causes a sudden stoppage of the milk secretions, complete loss of appetite, trembling and weakness to the verge of collapse, and sometimes pain manifested by constant treading. The pulse is rapid and weak, and the temperature may be normal or subnormal. In one case there may be no rise of temperature at any time, and in the next the fever may go to 104° F. within a few hours after the poison is taken. Convulsions and bellowing are almost pathognomonic of lead, when considered with the other usual signs. Fetid diarrhea is common, but not always present. In bracken poisoning there is an abundant bloody dysentery. The treatment of toxic gastroenteritis in animals offers little hope. After the poison is once absorbed, extensive damage to the liver and other organs commonly precludes recov-

ery. Large doses of magnesium carbonate in combination with the protective and stimulant treatment of enteritis may be of help in some cases.

Parasitic Gastroenteritis

Direct injury to the mucosa from parasites that suck the blood (*H. contortus*) or that develop as larvae within the wall (*Strongylidae*) may result in inflammation. The history reveals a chronic condition with gradual emaciation in which the symptoms of gastroenteritis may be either acute or chronic, usually the latter. Bloody diarrhea in calves a few months old may be caused by severe gastrointestinal infection with small strongyles or coccidia.

Traumatic Gastroenteritis

During the course of traumatic gastritis with peritonitis, cows may develop marked enteritis, though this secondary involvement is infrequent. It is marked by an abundance of mucus and thin watery feces, and may be confused with the primary form.

INFECTIOUS HEMORRHAGIC ENTERITIS IN SWINE

(Swine Dysentery; Bloody Dysentery)

Definition.—An acute highly fatal hemorrhagic inflammation of the cecum and colon affecting chiefly young swine and characterized by bloody dysentery.

In 1944 Doyle[1] reported the finding of a vibrio in association with swine dysentery, and this has been accepted as the cause.

Etiology.—Swine dysentery occurs chiefly as an enzootic among pigs and feeder hogs in the Middle West. Nearly all outbreaks are traced to the addition of swine purchased from stock yards or community sales. Within a week to ten days after arrival diarrhea appears, and within a few weeks it spreads through the herd and is transmitted to the breeding sows and other swine raised on the farm. It is most prevalent among the young, especially at weaning time. It may become endemic on a farm, affecting successive crops of pigs over a period of years. Hofferd[2] writes that in 1918 it was encountered in eastern Iowa in feeding hogs shipped into public yards; that it has spread to all parts of the State, and that losses up to 1,500 animals may occur in an area covered by a single veterinarian. Often it follows vaccination against hog cholera, a diet of corn, or the addition of either swine or cattle to the herd. According to Doyle,[3] "the causative agent is present in the colon and in the bowel discharge of affected animals. It is

possible to infect healthy swine, particularly young ones, by feeding small amounts of colon contents or bowel discharge from a dysentery hog. Thus far we have not been able to reproduce it by feeding viscera other than the colon, although such viscera may contain large quantities of suipestifer." In 1924 Whiting[4] reported that in his work on dysentery he was able to transmit the disease experimentally and consistently by feeding colon contents and feces from affected swine.

It seems to spread through the contamination of feed by sick hogs and usually it does not spread to other farms except through the transfer of exposed and infected hogs, or exposure to drainage from infected premises. Yet the disease may occur in herds to which hogs from other sources have not been added, and some believe that it may be introduced by visitors and venders. *Salmonella suipestifer* frequently occurs in the tissue of hogs that have swine dysentery, as it does in those affected with hog cholera. Its presence has no diagnostic significance, and the disease which it produces when fed in cultures does not closely resemble swine dysentery. Microscopic examination of scrapings from the cecal or colic mucosa, as well as the bloody mucus of the bowel discharge, usually shows the presence of large numbers of vibrios—Doyle.[6]

Morbid Anatomy.—When pigs are killed and autopsied in the early stages of the disease the cecal and colon mucosa show congestion, hemorrhage, increased secretion of mucus, and the colon often contains blood. Later there is sloughing of diphtheritic exudate that may be mixed with the contents of the colon. The lesions in the colon are always present, and closely resemble those of hog cholera. A gastritis is often found, but the small intestine is usually normal. The ileocecal junction sharply separates the normal from the diseased tissue. When viewed from the serous surface the wall of the colon may show diffuse reddening.

Symptoms.—As reported by Doyle,[3] in field outbreaks the shortest period of exposure was 7 days and the longest 60 days; in experimentally infected animals the incubation period varies from 7 to 12 days. As a rule the diarrheal symptoms appear on the 7th to the 9th day following the feeding of infectious material. Scouring in a few hogs is the first symptom, and usually there is a history of deaths. Early in the outbreak only a small percentage of the animals are sick; often there is a record of arrivals within one or two weeks, and a few weeks later the affection is general throughout the herd. At first there are dullness, lack of appetite and a rise of temperature that may go as high as 106° F. In many cases the temperature may be found only slightly above normal, and with the development of diarrhea the fever tends to sub-

side. In severe cases the animals rapidly become gaunt, weak, and show great prostration. The feces are usually mixed with blood and mucus and are red in color; in older animals the feces are darker or chocolate colored ("black scours"), and occasionally hogs die without showing symptoms of diarrhea. The course is from a few days to about two weeks; it is shorter in mature hogs; in the young recovery may be incomplete. The symptoms of the chronic form are diarrhea, emaciation, and stunted growth. The mortality in pigs is from 40-50 per cent; in feeder hogs, from 10-20 per cent; and in brood sows, 2-5 per cent.

According to Hofferd[2] a corn diet may set it off after it is under control. It may closely resemble either hog cholera or swine erysipelas and differential diagnosis may be difficult, especially in the beginning. Bloody feces are the most distinctive symptom. The difficulties involved are expressed by Bryant[5] in the statement that "when it is found that hog cholera is complicated with other infections, more especially swine dysentery, the practitioner faces a greatly involved and alarming situation." He expresses the view that "shoats raised in contact with yard infection, of low virulence, build a resistance against that infection which may tide them over the serum-virus reaction, while thrifty appearing hogs having no acquired resistance to enteritis become easy prey during the serum-virus reaction."

Control.—The method of control is to remove all animals from contaminated lots and houses. Isolate the sick and place on an easily digested limited diet. The use of bacterins or vaccines is of doubtful value. Hofferd[2] suggests the use of serum alone when both swine dysentery and hog cholera are present in the herd; his method of procedure is also to provide sanitary surroundings, fresh drinking water medicated with guaiacol compound, and feed only soaked oats treated with alkaline mixture. A diet of ground oats and buttermilk is especially recommended. In many cases the most satisfactory and effective way to handle dysentery is to sell all the hogs for slaughter and then start over with clean yards or pastures with animals from a healthy source.[6]

Antibiotics. In 1951 Salisbury[7] et al reported curative action from streptomycin: 1 Gm. per pig under 100 lb. of body weight, and 1½ Gm. per pig up to 150 lb. and succeeding doses of ½ the initial dose every twelve hours for six and one-half days; this was mixed in the feed or dissolved in 40 cc. sterile distilled water and given by dose syringe. Curative action also followed the use of aureomycin: initial dose of 25 mg. per pound of body weight mixed in the feed or dissolved in 40 cc. sterile distilled water administered with a dose syringe. The maintenance dose was ½ the initial dose and was administered every twelve hours for four and one-half days. "In all cases there was an improve-

ment in the character of the feces of the treated animals within forty-eight hours and a return to normal in three to four days after the start of treatment, regardless of the antibiotic used."

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INFECTIOUS NECROTIC ENTERITIS

(Salmonellosis; Infectious Enteritis; Pig Typhus; Paratyphoid; Infectious Suipestifer Enteritis)

Definition.—A severe diarrhea in pigs and young swine that on autopsy show necrotic lesions in the cecum and colon. *Salmonella choleraesuis* may be recovered from the diseased tissues, and it is regarded by most authors as the primary cause of the disease. Necrotic enteritis is reported by Kernkamp[1] to be the most important gastroenteric disease of swine, affecting as high as 20 per cent or more of the herd. Suipestifer may also manifest itself as an acute septicemia—Van Es,[2] Seghetti.[3]

Etiology.—In 1929 Murray[4] and associates reported that they had isolated suipestifer from 100 per cent of field cases of infectious enteritis, and had fed virulent cultures to more than 100 head of young swine with 100 per cent infection in the experimental cases; they were unable to recover it from the intestines of normal swine. In cases destroyed early, suipestifer was always found in abundance. Associated with this infection they always found *Actinomyces necrophorus,* which they regard as a secondary invader; in cases destroyed early, *A. necrophorus* is either absent or scarce, while it is abundant in the later stages of the disease. Other susceptible experimental animals are mice, rabbits, and guinea pigs. The immunity produced by feeding is short-lived and slight. In the presence of ascariasis the intensity of the attack is increased.

As described by Biester and Murray,[5] "*S. choleraesuis* produces the initial injury and appears in greatest numbers near the surface and outer portions of the caseated membrane. These decrease in number

towards the inner portions while necrophorus organisms appear in greater numbers, so that upon reaching the base of the lesion only necrophorus-like organisms are found upon direct histologic examination. . . . In cases where the caseated zone is deep and has been present for a long period the necrophorus bacilli are very numerous. In chronic cases the destruction of tissues by the necrophorus organism overshadows everything else, so that it should not be considered merely as an indifferent secondary invader, but as an important factor in this form of enteritis. . . . Often the erosion involves the vessels before complete clotting takes place and blood passes into the intestinal lumen." Since an attack does not confer immunity, the disease may recur.

Ever since Salmon and Smith[6] reported *B. suipestifer* to be the cause of hog cholera in 1885 its relation to diseases of swine has been a confusing problem. With the discovery in 1903 that hog cholera is caused by a virus, suipestifer came to be regarded as a secondary infection responsible for necrotic lesions ("button ulcers") sometimes found in chronic cholera. It is now recognized as an occasional primary cause of acute septicemia in swine, especially in cholera-free areas. There are numerous field reports that the use of virus in pigs susceptible to cholera appears to initiate an outbreak of necrotic enteritis after an interval of from ten to thirty days. And Doyle[7] writes that "in producing hyperimmunity in swine by injection of increasing amounts of hog-cholera virus it has been observed that certain individuals develop diarrhea, become unthrifty, and present the symptoms and lesions of necrotic enteritis; such individuals may eventually fail to show the presence of virus in the blood." An example of the justified dread of this disease following vaccination is shown in a report by Bryant[8] of 25 deaths from necrotic enteritis after vaccination of 250 apparently normal shoats that may have been exposed to cholera before vaccination. Furthermore, it may be difficult or impossible in the field to differentiate necrotic enteritis from hog cholera (Tischhauser)[9] or swine erysipelas (Van Es).[2] It is known that *Salmonella choleraesuis* may be a secondary invader in hog cholera and it is widely credited as the primary infection in necrotic enteritis.

The influence of unfavorable sanitary and nutritional conditions is universally recognized and is emphasized by Bryant[8] who writes that pigs from two to four months old may well be expected to develop the classical symptoms of necrotic enteritis, as persistent diarrhea, unthriftiness, and variable appetites when the farms abound in mud and filth holes, and it is on such farms that the disease usually occurs. Overfeeding, especially from self-feeders, is often reported as a cause of enteritis in pigs.

Morbid Anatomy.—The lesions are chiefly localized in the stomach, cecum, colon, and rectum, the small intestine being less frequently involved. The most marked changes and distinctive lesions, however, are found in the cecum and colon. The walls are greatly thickened and the mucosa is diffusely covered with a yellowish gray layer of necrotic, caseated tissue. On removal of this diphtheritic layer the underlying mucosa is found to be diffusely red and granular. In some cases the necrosis is in the form of patches. The epithelium is extensively destroyed and the changes may extend to the submucosa. The necrotic tissue may slough off and be mixed with the intestinal contents. In acute types there may be merely a catarrhal exudate, or small hemorrhages and edema of the mucosa. Similar changes may involve the small intestine, especially the lower part. The gastric mucosa may be normal, or present lesions varying from hemorrhage to the formation of ulcers. The lymph glands, especially those of the digestive tract, are swollen, congested, and edematous. As described by Breed,[10] "The spleen is enlarged, swollen and dark. The kidneys occasionally show hemorrhages on the surface which are large, dark and irregular. On the cut surface large, dark hemorrhages are quite constant in the papilla of the medulla."

Symptoms.—The onset is usually sudden in pigs from 2 to 4 months of age, though scours and unthriftiness may appear as early as 2 weeks. The bowel evacuations often contain fibrinous or necrotic tissue, and in some cases blood. As a rule the temperature is high and the appetite is variable. After a few days the temperature may return to normal and the appetite improve, but the animals remain unthrifty and fail to gain weight. When no efforts at control are made, emaciation, prostration and death are frequent. During the early stages it may be diagnosed as hog cholera, and if this is followed by the administration of hog cholera virus and anti-hog-cholera serum combined the condition becomes worse and many deaths occur. The course may be either acute or chronic and the mortality is high.

The relation of nutrition to enteritis in pigs has been reported by several observers. In 1939 Madison et al.[11] reported an outbreak of nicotinic acid deficiency in swine in the field. Nicotinic acid checked the mortality and restored the remaining swine to health. In 1940 Davis et al.[12] described a deficiency disease in young swine on experimental single-grain feeding. The symptoms were unthriftiness, watery diarrhea, loss of weight, and a wrinkled scurfy skin. This syndrome appeared chiefly in pigs on a diet of yellow corn and occasionally in those fed oat groats. The grain ration was supplemented with 1.5 per cent mineral mixture consisting of equal parts steamed bone meal,

ground limestone and salt together with a small amount of ferrous sulfate and potassium iodide. A maximum of 6 lbs. of buttermilk was given daily to each pig, and carotene in oil or cod liver oil concentrate was fed regularly to supply additional vitamin A. The majority of cases appeared in animals that weighed between 30 and 60 pounds, the period of symptoms was from 30 to 60 days or more, and deaths were frequent. The autopsy lesions were those of necrotic enteritis limited to the mucosa of the colon and cecum. In a few chronic cases there was a secondary pneumonia. The administration of nicotinic acid (60 to 100 mg. daily), fresh liver (200 Gm. daily), or yeast and liver meal was followed by rapid recovery. These results justify a diagnosis of pellagra, and suggest a possibility that nicotinic acid deficiency may be a factor in some field cases of swine enteritis. It may be more than a coincidence that experimental pellagra has been caused by a corn or oats diet, and greater frequency of enteritis has been reported by Bryant[8] in swine on a corn and oats diet. The term "pellagra" is from the Italian "pelle agra," meaning rough skin, and the pellagra preventive factor is vitamin B_2, a substance containing nicotinic acid, riboflavin, and vitamin B_6. It has been shown that nicotinic acid is a curative agent in swine. In 1942 Edgington[13] and associates reported that the protective value of nicotinic acid is not sufficient to encourage its use as a preventive or curative measure.

In considering the various causes of infectious enteritis, the distinguishing features of pellagra are the gradual onset, chronic course, scurfy eruption of the skin, absence of fever, and lesions confined to the gastrointestinal tract.

Control.—The method of control is to remove all animals from contaminated lots and houses, isolate the sick, and place on an easily digested limited diet. The use of bacterins or vaccines is of doubtful value.

In the treatment of both acute and chronic forms of enteritis, including bloody dysentery, Kernkamp[14] obtained 69 per cent recoveries in 36 treated swine, and 16 per cent recoveries in 31 controls from the administration of sulfaguanidine 0.16 to 0.33 Gm. per kg. body weight (or 0.75 to 1.5 Gm. per 10 lb. body weight). In several lots 166 were treated under field conditions with 92 per cent recoveries. Similar results were reported by Cameron,[15] who gave as high as 1 Gm. per 20 lb. body weight four times daily; suckling pigs received 1 Gm. three times a day. Similar results have also been reported from the administration of sulfasuxidine, and sulfathalidine. Antibiotics, such as streptomycin or aureomycin, are indicated.

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COCCIDIOSIS

(Red Dysentery in Cattle; Hemorrhagic Coccidiosis)

Definition.—A specific hemorrhagic enteritis affecting the rectum, to some extent the colon and cecum, caused by coccidia (*Eimeria zurnii, E. bovis, E. ellipsoidalis*) and manifested clinically by the passage of thin feces usually containing fresh clots of blood.

Etiology.—Coccidiosis has been reported in cattle, swine, and lambs in widely separated parts of the United States, where it is far more frequent and important than is generally recognized. Extensive infection has been reported in range cattle in North Dakota, and U.S.B.A.I. Reports[1] of field studies in Alabama and Georgia have emphasized the prevalence of clinical and subclinical coccidiosis in calves. In New York it is found in many herds each year and it is increasing in both stabled and pastured animals. It is highly prevalent in cattle in Switzerland and Ireland and its distribution is world-wide. There are numerous reports from Colorado and Nebraska of its presence

among feeder lambs. A Bureau Report (1939)[1] gives a brief description of experimental infection in pigs, but natural infection in swine in this country is rare.

In its seasonal incidence coccidiosis is distributed throughout the year, but in each year it may be high in some particular month. In our clinic the favorite months have been from April to December, with the greatest number of cases in October. According to Marsh,[2] in the Northwestern States coccidiosis is typically a disease of young cattle, occurring most frequently in calves. The majority of our cases have been between the ages of 4 months and 2 years. It has been observed, however, in a calf only one month old and in a series of 20 cases seen in the ambulatory clinic from July to October, 1938, 13 were in cows between the ages of 3 and 9 years. *Eimeria zurnii* is the species of coccidia chiefly pathogenic to cattle, though ten different species with various degrees of pathogenicity have been described. Infection is by ingestion of oocysts, each containing four spores—*sporozoites*, in the food or water. On reaching the intestines these spores are liberated and they enter the epithelial cells. Here they first undergo a sexual multiplication where all stages of development may be found. Finally there is a process of fertilization with the formation of immature oocysts, each containing four spores and the parasite is then expelled in the bowel evacuations. The time required for this reproduction varies from one to four weeks. This development of the parasite in the epithelium causes destruction of cells over large areas and results in capillary hemorrhage, hemorrhagic enteritis, and bloody diarrhea. If the invading oocysts are few in number and the animal is well nourished, there may be no clinical reaction and the individual may acquire an immunity or resistance against subsequent infection. Observations indicate that all bovines harbor oocysts and that the vast majority acquire resistance. Immature forms of oocysts are expelled in the feces and on exposure to moisture and air they sporulate to maturity in two to three days. They are oval, sometimes round, varying from 30 to 35 microns in length by 20 microns wide. They resist freezing and disinfectants and they may remain viable outside the body for one or two years. They are destroyed by putrefaction, sunlight, and drying. Oocysts are abundant in the feces or rectal scrapings of the sick, but they may not be found in the feces in the beginning of an attack; they are also found in the feces of the well—carriers. It is possible, however, that oocysts found in "carriers" are nonpathogenic. It has been found impossible to raise animals free from contamination with oocysts. They are widely distributed outside the body, both on fields and in stables. Wherever fecal contamination is possible, oocysts may

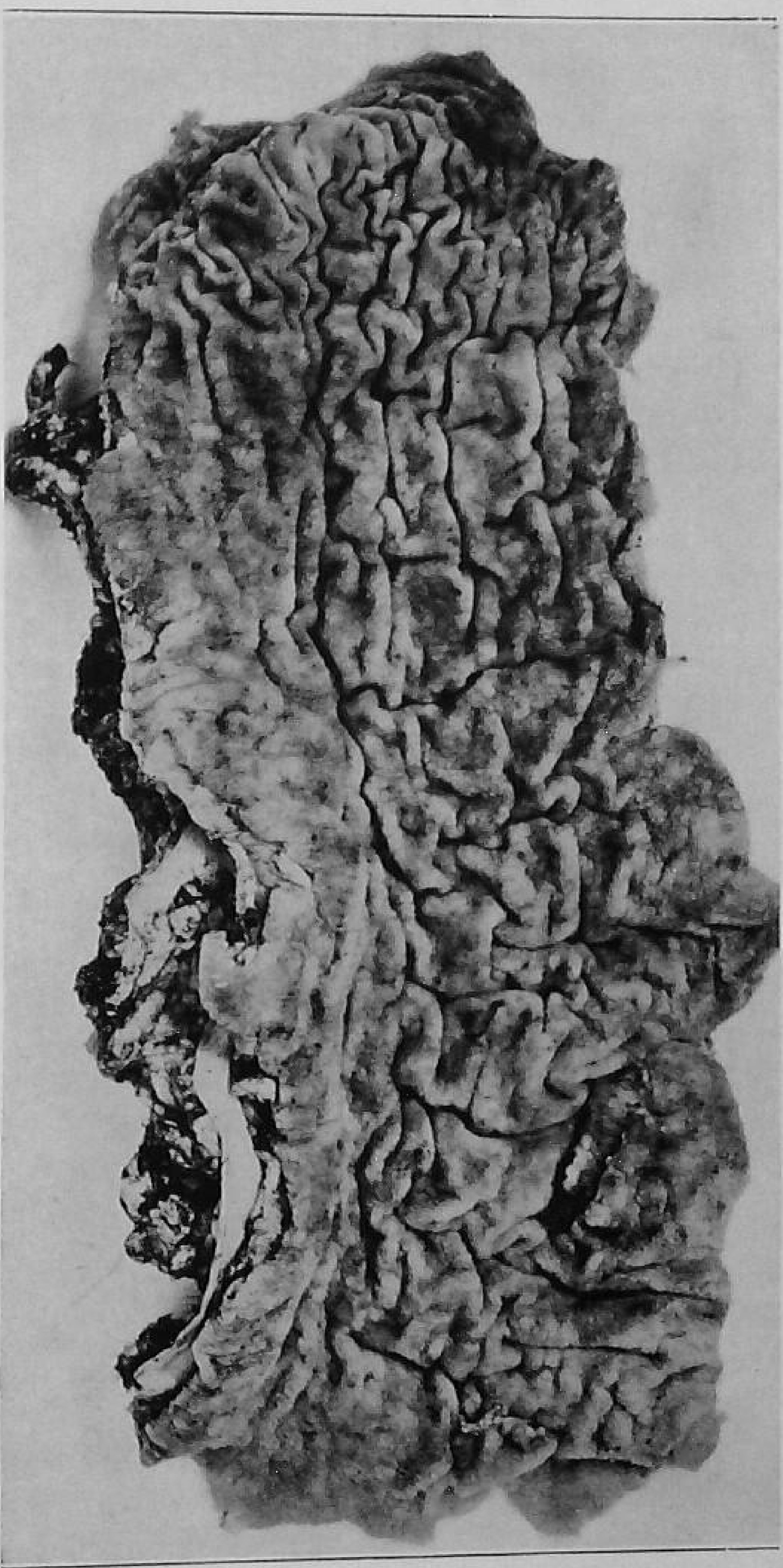

Fig. 20.—Coccidiosis, a section from the cecum showing thickening of the wall and corrugation (Way and Hagan), Cornell Vet., 1920, 10, 17.

be harbored. Each class of livestock harbors its own species, thus there is no cross-infection between cattle, sheep, and swine.

Mode of Infection.—It has been rightly claimed that infection is favored by filth, moisture, and poor sanitation, but it is not infrequent where the hygiene is excellent. With few exceptions the disease is either sporadic or mildly enzootic. The infection may attack a single individual in a remote neighborhood where there has been no contact with cattle that drink from a common stream. Lamont[3] has reported that in Ireland it is as common in dry as in wet seasons and is probably caused by drinking from stagnant pools. It is generally believed that the number of oocysts ingested determines whether or not the disease will develop. In enzootics of coccidiosis it is a common observation that the cattle drink from surface water, often they are poorly nourished, and occasionally they are housed in damp filthy stables.

In an extensive outbreak of coccidiosis in feeder lambs in Nebraska, it was observed that the lambs were infected on arrival, that deaths and scouring and heavy discharge of oocysts began after twelve to sixteen days of feeding, and that death loss ceased after symptoms had lasted for about two weeks; corn silage fed in open troughs that were never cleaned provided ideal moisture conditions for the sporulation and preservation of the viability of oocysts in the fecal pellets that lambs carried to the feed—Christensen.[4]

Morbid Anatomy.—The cadaver is emaciated and the tissues are anemic. The posterior parts of the body are often smeared with bloody feces. Hemorrhages may be found on the pericardium and pleura. The most constant and characteristic changes are in the cecum, colon and rectum; their mucous membranes are thickened, hemorrhagic, often edematous, and frequently they contain clots of blood. The mucosa of the rectum is highly reddened and hemorrhagic presenting thick longitudinal folds; the wall is thickened to two or three times the normal. The contents of the terminal portion of the colon and the rectum may consist largely of blood. Occasionally there is hemorrhagic inflammation of the mucosa of the abomasum and the small intestines.

Symptoms.—The period of incubation is from one to three weeks. In a mild form bloody dysentery and a lessened milk flow are the only symptoms. In suckling calves there may be only a slight diarrhea with bloody feces containing many oocysts. In more severe forms there is an early loss of appetite associated with rapid loss of condition, weakness, and fever, though the temperature may be normal or subnormal. The pulse is fast, 80 to 120, and the mucous membranes are pale. An early distinctive symptom is watery, fetid, bloody dysentery with tenesmus and the passage of fresh clots of blood. In fatal attacks the bowel

evacuations may be almost clear fluid blood. Peristalsis is increased at first but it may be followed in one or two days by complete paralysis of the bowels. The rectal mucosa is thickened, congested, and wrinkled; the rectum may remain open, and the hind parts are smeared with bloody feces. Pneumonia is frequent in calves and recovery from coccidiosis in calves may be followed by retarded growth.

A blood examination discloses the extreme degree of anemia. In one very sick 2-year-old Jersey heifer which recovered a blood report was as follows:

Red blood cells	2,110,000
White blood cells	7,900
Hemoglobin	35%
Lymphocytes	45%
Neutrophiles	55%

Towards the end of a fatal attack nervous symptoms often appear, such as twitching of the muscles, delirium, and paresis until the animal staggers and falls with legs spread apart and beyond control.

The course is from three or four days to two weeks. In fatal attacks death often occurs on the fourth or fifth day, and the first to succumb may die even earlier—one or two days. The mortality in a series of cases treated in the ambulatory clinic in 1945-50 has been less than 10 per cent. In a stable enzootic of poorly nourished cows or young stock the mortality may be much higher. In a report by Gibbons and Baker[5] there is a record of 3 deaths in a herd of 31 in which all were affected except 5 calves aged 3 to 4 months. All except the calves drank from a shallow stream which was regarded as the source of infection; this was an undernourished stabled herd in December, 1937. In a second outbreak in July, 1938, there was a mortality of 50 per cent of 16 cases divided equally between young stock and cows; the course was from four to seven days. There was definite proof of spread of infection to six farms by surface drainage.

As described by Simms and associates[6] in Georgia and Alabama, coccidiosis not only presents the classical type of infection characterized by bloody scours, but also a more prevalent nonbloody subclinical coccidiosis. Persistent diarrhea occurs in calves from one to three months old. Very few calves less than three weeks old show oocysts, but older calves may show an extent of infection often found in clinical cases. In general there is less trouble in herds kept under sanitary conditions.

Diagnosis is based on the amount of blood in the feces, the high mortality, the postmorten changes, and the presence of many oocysts in the feces. While oocysts are generally found on microscopic exam-

ination of feces, they may be absent in the beginning of the attack, and they may not be found in the feces a few weeks after recovery. One should not always exclude a diagnosis of coccidiosis, therefore, when an examination of the feces is reported negative. Two or three days later the oocysts may be present in large numbers. The fact that a few oocysts may be found in the feces of most normal animals indicates that the number, and perhaps the species, rather than the presence alone is of chief significance. The symptoms in a severe outbreak of winter dysentery may closely resemble those of coccidiosis. The differential features include regard for a possible source of infection; frequently coccidiosis is associated with drinking from shallow pools or shallow contaminated streams. When winter dysentery is prevalent there are usually a number of affected herds in the area and the seasonal incidence is from December to March. While our experience has been limited, only 7 of 90 cases of coccidiosis have been observed in the months of January to March: 3 in February, 4 in March, and none in January. In severe winter dysentery with bloody diarrhea there are rarely any deaths, and oocysts are not present in sufficient numbers to establish a diagnosis of coccidiosis. In calves one to three months of age hemorrhagic enteritis of the small intestine may cause bloody diarrhea similar in appearance to that of coccidiosis.

A characteristic lesion in the posterior part of the small intestine of especial diagnostic value in cases which come to autopsy in the early stages of the disease, prior to oocyst production, has been described by Boughton.[7] The posterior half of the small intestine is found sprinkled with small, white, cyst-like dots pinpoint in size. Several of these bodies may be enmeshed in the capillary network of a single villus. They are believed by Boughton[8] to be large asexual stages (schizonts) of one or more species of bovine coccidia, *Eimeria bovis* being the most likely one. Fresh smears from mucosal scrapings may reveal masses of developing oocysts.

Treatment.—Many drugs have been used for the treatment of coccidiosis, and in the majority of instances the action sought has been that of an astringent or protective to the intestinal mucosa. If the attack is mild, recovery occurs in a few days without treatment; if it is severe some deaths are bound to occur for the mortality is high. In the handling of severe attacks the use of protectives for the intestinal mucosa needs to be supplemented by stimulants, by the use of normal saline solution and blood transfusion or citrated blood to combat dehydration and hemorrhage, and of dextrose to support nutrition. Astringents and protectants are represented by tannic acid (1 ounce) and bismuth subnitrate (1 to 4 ounces in warm milk). Mineral oil (1 quart

daily) is useful to remove fetid intestinal contents and it is also a protective. In severely sick animals, improvement has followed intravenous injection of 500 to 1000 cc. of citrated blood, and intravenous injection of 1000 to 2000 cc. of normal saline solution containing 5 per cent dextrose. Severe tenesmus may be relieved by rectal injection of one quart of warm mineral oil; retention and relief are favored by epidural anesthesia.

In the treatment of coccidiosis in young stock one may give from 30 to 45 Gm. of sulfaguanidine daily to animals 10 to 12 months of age. Sulfathalidine may be combined with either sulfamerazine or sulfamethazine. As described by Fox and Roberts,[9] 1¼ grains of sulfathalidine and ¾ of a grain of sulfamerazine or sulfamethazine per pound of body weight were given orally for two or three days. In extremely emaciated individuals a supplemental vitamin-mineral supplement containing dry yeast was given for a period of 2 to 4 weeks. It is believed that sulfathalidine attacks the coccidia on the mucosa, and either of the others attacks the coccidia in the tissues. This combination appears to be the most effective treatment now known.

Prophylaxis is most effectively accomplished by protecting the water from surface contamination, by providing dry well-bedded quarters for young stock, and by adequate feeding. For the numerous sporadic scattered infections there is no effective protection, since there is no way of knowing when or where a susceptible animal will acquire a massive dose of oocysts. The disinfection of stables at the time of an enzootic is of doubtful value; in such instances the source of infection may be found in the water supply. For the destruction of oocysts, ordinary disinfectants are ineffective. They are destroyed by drying at ordinary temperature and by heat above 40° C. According to experiments conduced by Enigk[10] oocysts were destroyed in 20 seconds on exposure to a 2 per cent solution of carbon disulfide containing 2 per cent phenol. For cleaning stable floors and walls previous to disinfection, a "steam jenny" with a solution of hot lye under pressure will loosen 2-3 inches of manure from the floor and thoroughly clean the walls and ceiling.

In an experiment reported by the Bureau of Animal Industry (1941) to test the prophylactic and therapeutic effect of sulfaguanidine against natural subclinical infections of young lambs, sulfaguanidine was administered in 2-gram doses daily except Sunday. It prevented completely the acquisition of infection in five lambs and rapidly reduced heavy existing infection in four lambs. Simms[6] was able to prevent the disease in calves by segregation according to age in the following groups: 1, under three weeks old; 2, three to six weeks; 3, six weeks to three months; and 4, over three months.

"At the United State Regional Animal Disease Research Laboratory, Auburn, Ala., a portable pen 5 by 10 by 3 feet in size was found to be of value in controlling coccidiosis in dairy calves. Records kept over a period of years showed that when young dairy calves were not adequately protected against coccidiosis and other diseases, such as white scours and pneumonia, up to about 75 per cent of the animals died. For several years the movable pens have been tested with 177 calves on the laboratory grounds, and during this time the mortality was only about 11 per cent from all causes. The only labor involved is lifting the pens each week and moving them uphill to a fresh site. This can be done easily by two men. The low mortality is remarkable for the region where this work was done, especially since the calves were deliberately inoculated with coccidia. In Alabama alone, over 300 of these pens were in use during the year on about 80 farms. Dairymen using them reported calf losses of less than 10 per cent on farms where the losses had been up to 75 per cent prior to their use."[11]

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ANIMAL PARASITES IN THE STOMACH

GASTROPHILUS LARVAE IN THE STOMACH OF THE HORSE

(Ascariasis)

Three species of the genus *Gastrophilus* infest American horses: (a) *Gastrophilus intestinalis (equi)*—horse botfly, common botfly; (b) *G. nasalis (veterinus)*—throat botfly, chin fly; and (c) *G. hemorrhoidalis* —nose fly. The larvae of these flies are termed bots.

Life History.—In the Northern States botflies appear in early summer, May and June, and become especially abundant and troublesome in the latter part of the warm season. The common botfly is reported from practically all parts of the United States; the throat botfly is widely distributed; the nose fly has a more limited distribution and is mostly confined to the North Central States and certain of the Rocky Mountain States, but it is rapidly spreading in all directions—Schwartz.[1] The eggs of the common botfly are deposited on the hair of the forelegs, shoulders, and chest wall. The eggs of the throat botfly are deposited on the long hairs in the intermaxillary space, while those of the nose fly are laid on the hair at the edges of the lips. The larvae of the common botfly gain entrance to the mouth when the horse brings its lips into contact with the hairs on which the eggs are placed. Hatching occurs in not less than seven days and it may be delayed for as long as three months. The eggs of the throat botfly hatch without friction or moisture; Bishopp and Dove[2] report that they have never found living larvae within them. Upon hatching the larvae crawl through the hair and enter the mouth. According to Wells and Knipling[3] the larvae of the nose botfly pentrate the epidermis of the lips and migrate in this tissue into the mouth. *G. intestinalis* usually lives in the esophageal portion of the stomach, while *G. nasalis* and *G. hemorrhoidalis* usually live in the region of the pylorus—Hall.[4] On reaching the stomach, the larvae attach themselves to the mucosa where their development is completed; after eight to twelve months they become detached and pass out with the feces. In their migration through the intestines, larvae of the nose fly may attach themselves to the rectum or anus. On reaching suitable soil, as in a plowed field, the larvae of the botflies burrow just beneath the surface, pass through the pupa stage, and reappear as flies in from three to ten weeks.

Symptoms.—Botflies are of considerable importance, first because

of annoyance and fright caused by the attacks of the flies, and second, because of irritation or obstruction in the stomach and pylorus. Attacks of the chin fly cause the horse to toss the head violently and to stop when at work and try to place its head over the neck or back of its mate. Attacks of the nose fly cause the horse to become terrified and unmanageable. There has been some difference of opinion in regard to the injury caused by bots; it is probable that they are harmful to young

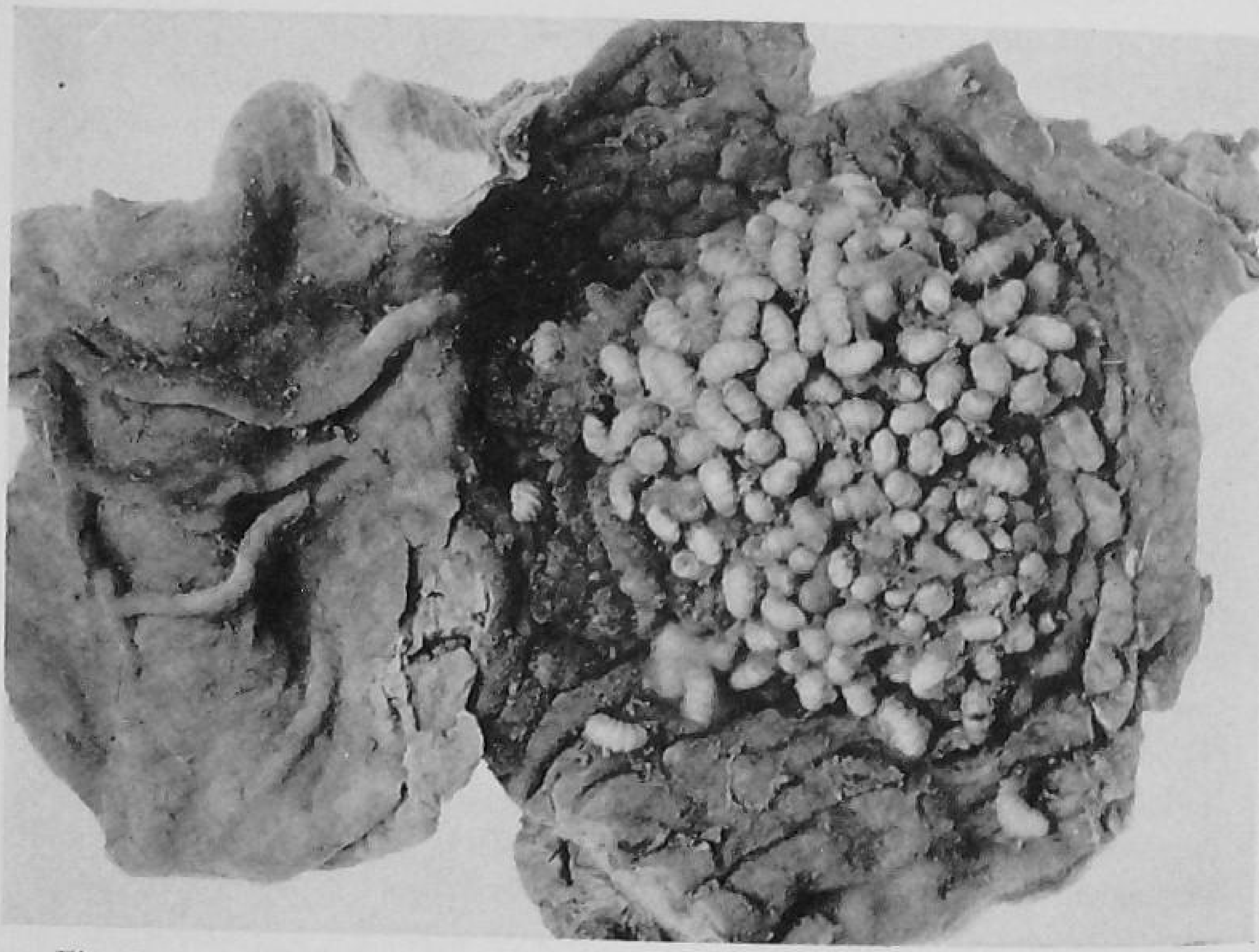

Fig. 21.—Gastrophilus larvae in stomach of the horse at the entrance to the duodenum. These parasites caused dilatation and rupture of the stomach as shown in Fig. 22.

animals when present in large numbers. A report by Todd and Doherty[5] on surveys made in Kentucky indicates that ascaris infection is largely a problem of sucklings, weanlings, and yearlings; no heavy ascaris infections have been found in mares, and such infections are rare in older animals due to an apparent age immunity. As in other forms of intestinal parasitism in the horse, there are unthriftiness in the winter, pale mucosa, and irregular appetite. In making a diagnosis, one considers the history with respect to eggs on the hair during the summer, and the degree of infection with other parasites as revealed by the eggs in the feces. Since carbon disulfide is effective in the expulsion of bots, its use in diagnosis is helpful. Bots may enter the bile ducts, caus-

ing fatal occlusion. The symptoms are severe colic, extreme jaundice, convulsions, and death within a few hours. I have observed two fatal cases from the accumulation of bots in the pyloric region of the stomach and duodenum; one caused gastric dilatation with rupture after a course of less than twenty-four hours; the other caused death from rupture of the duodenum after an attack of severe colic lasting about forty-eight hours; both were in the month of June.

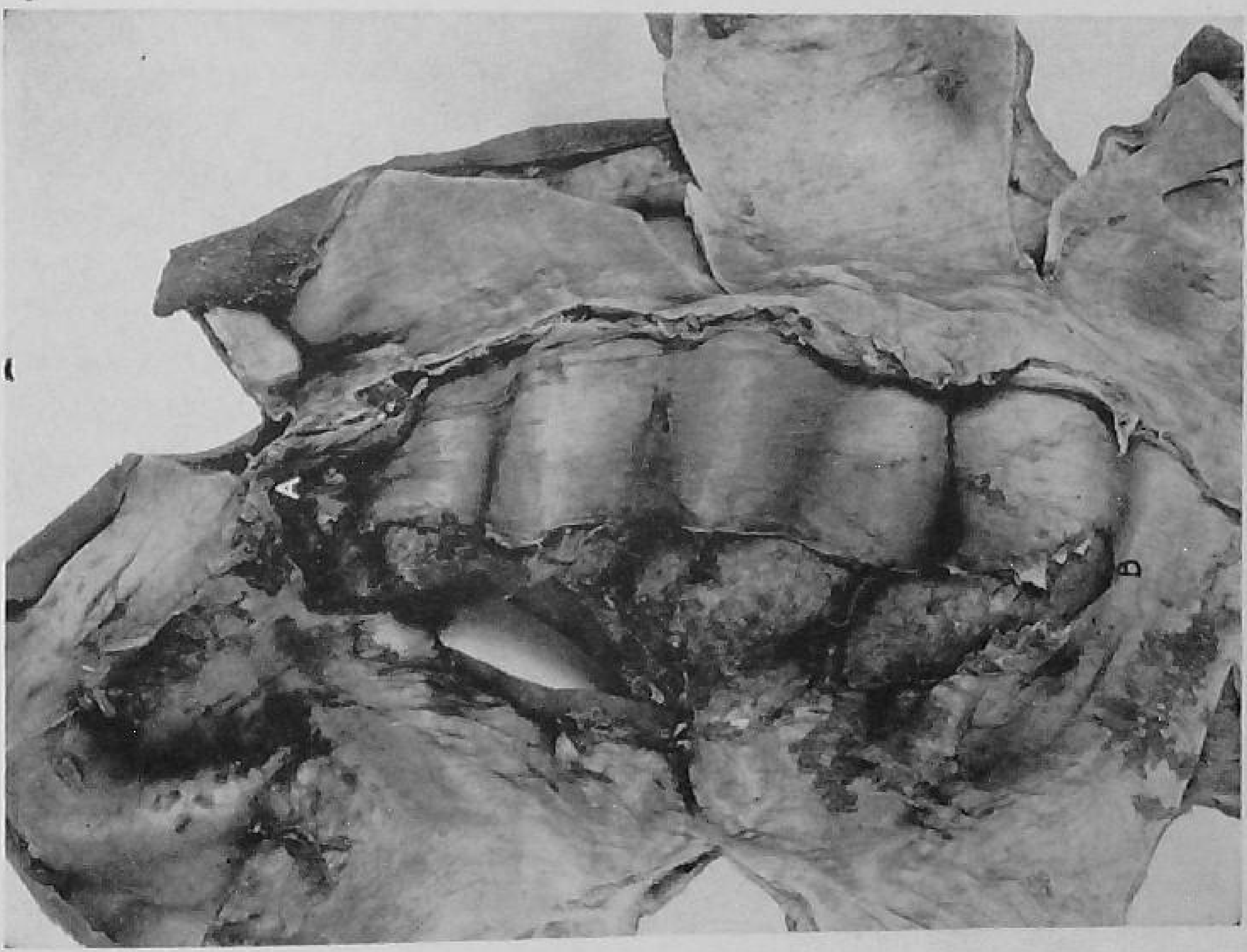

Fig. 22.—Gastric rupture seen from the peritoneal surface. *A* to *B*, tear in the peritoneum showing hemorrhage, and a much smaller opening through the mucosa.

Treatment and Prophylaxis.—According to Hall,[4] carbon disulfide is 100 per cent effective in the expulsion of bots, and it is also effective in the expulsion of ascarids. Withhold food from noon until after medication the following forenoon; give water the previous evening, but do not feed or water on the morning of the day of medication. The carbon disulfide may be administered in liquid form through a stomach tube followed by about 2 ounces (60 Gm.) of water, or given in a mass capsule. Some prefer the liquid form because of serious injury that has occasionally resulted from lodgement of a capsule in the throat. The dose is 6 drams (24 cc.) for mature horses and 3 drams (12 cc.) for colts; estimate 1.5 drams (6 cc.) for each 250 pounds (125 kg.) of weight.

After medication with carbon disulfide, no laxative should be given, and both food and water should be withheld for three hours.

Todd et al[5] attained the highest elimination of ascarid eggs from foals when treatment was commenced on June 30, at an average age of 13.4 weeks, with a dosage of 4 to 6 drams of carbon bisulfide administered through a stomach tube, and repeated on Aug. 10 and Aug. 24 with a dosage of 5 to 6 drams. The yearlings were given a single treatment of 6 drams.

Its use in mares in advanced pregnancy is not advisable, and caution should be observed in the medication of colts, old horses, and those that are in poor condition or weak. Carbon disulfide irritates the mucosa and it may cause symptoms of colic. Eggs that are still present on the hair at the time of medication contain latent larvae and should be removed. According to Wells and Knipling[3] the larvae are not affected by disinfectants, but are destroyed by "sponging copiously the infected portions of the coat with water at 104° to 118° F. on a day when the air temperature is below 60° F. This treatment decoys the waiting larvae from the eggshell, after which they die from exposure without gaining entrance to the mouth of the horse." Where only one treatment is given for bots in horses it should be administered early in February in the Northern States, and late in February in the Southern States. When two treatments are given the first may be administered early in December. Weekly removal of the eggs from the hair will prevent the entrance of larvae into the stomach. Carbon tetrachloride and tetrachlorethylene are also fairly effective against bots.

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STOMACH-WORM DISEASE OF SHEEP AND GOATS

(Strongylosis; Haemonchosis; Wireworm Disease; Trichostrongylosis)

Definition.—A chronic catarrh of the abomasum characterized by diarrhea, anemia, and emaciation, caused by *Haemonchus contortus, Trichostrongylus axei,* and *Ostertagia circumcincta.* These parasites abstract nutrition by sucking the blood; they injure the mucosa and cause anemia. In association with stomach-worm disease one often finds a similar affection of the small intestines, also caused by various kinds of round worms. According to Fourie[1] hemolysins are not formed by *Haemonchus.* While the method of attack of the different genera is somewhat variable, this variation has little practical significance since the infection is usually mixed.

Etiology.—In all sheep-raising countries stomach-worm disease is one of the most destructive affections. It is common throughout the United States, especially in parts of the Middle West and South where it has caused heavy loss. In permanent pastures it is often fatal to lambs, and in heavy invasions to older animals as well. Stable-fed lambs may also become infected. Often it occurs with other parasitism, such as nodular disease, lungworm disease, and parasitism of the small intestines. In the eastern and southern parts of the United States where there is an abundance of moisture, *Haemonchus contortus* has been the chief cause. According to Ross and Gordon[2] *Haemonchus* is rarely established in areas having an annual rainfall of less than 20 inches. It has been reported from California by Freeborn and Stewart[3] as a serious affection in both the southern and northern sections of the State, particularly from high, well-watered valleys and irrigated pastures. In the dry range country of the West, and on the Pacific Coast, *Trichostrongylus* and *Ostertagia* have been the chief cause of stomach-worm disease in sheep, but now they are also widely prevalent in sheep in the eastern part of the United States. The level of nutrition has a profound effect on the resistance of lambs to worm infestation—Fraser.[4]

Life History.—*H. contortus* is a slender round worm from 0.5 to 1.5 inches (1.27 to 3.81 cm.) long; its life history has been described by Ransom,[5] and Veglia.[6] The female is longer than the male and presents spirally shaped lines that represent the uterus. Thousands of worms may occupy the abomasum, where the eggs are deposited in the form of elongated oval bodies 75-100 microns long by 40-50 wide; these are present in large numbers in the feces. After leaving the body, they hatch in from fourteen to twenty-four hours under proper heat and moisture. In from one to two weeks after the passage of the eggs the parasite is infective; it is now in the sheathed stage and is resistant to

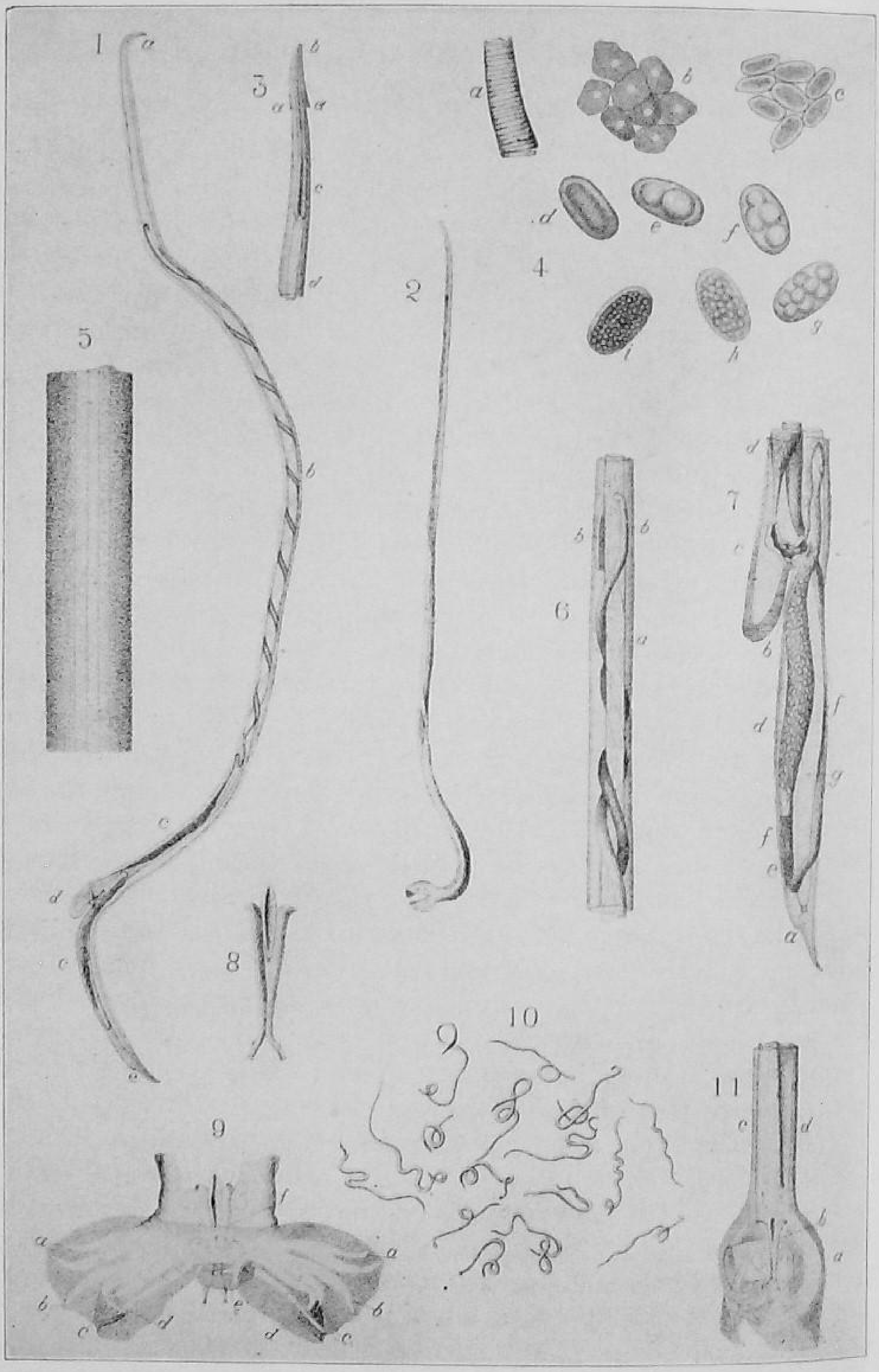

Fig. 23.—*Haemonchus contortus*. 1, adult female; 2, adult male; 3, head; 4, eggs; 5, skin; 7, caudal end of female; 8, spicula; 9, male caudal pouch; 10, group of adult males and females, natural size; 11, caudal end of male. (From *Parasites of Sheep*, Bureau of Animal Industry.)

dryness and cold; it is 0.6 to 0.8 mm. in length and moves actively on moist grass blades but does not feed. In *two to three weeks* after the sheep eats grass or other forage carrying the sheathed form, the parasite reaches maturity in the stomach. Infection occurs most readily in fields that have been used continuously for sheep pastures, and in wet seasons. Suckling lambs may become seriously infected in a few weeks by merely associating with infected dams in fields and lots not previously occupied by sheep, especially when the feed and water troughs are shallow and exposed to fecal contamination.

In the 1938 Report of the Bureau of Animal Industry[7] it is said that "Under outdoor conditions at Beltsville the infective larvae of the common sheep stomach worm, the sheep threadworm, and *Trichostrongylus* spp. died for the most part during the winter months. . . . These facts indicate that the survival on pastures of the larvae named is not nearly so important a factor in the carrying over of these nematode infestations from season to season as the persistence of the worms in the stock." Adults live in the stomach for several months; some were found by Ransom[5] a year and a half after the sheep had been removed from the pasture. Since the sheep carry the parasite when apparently normal, every animal may be regarded as a source of contamination.

According to Ross,[2] where sheep do not succumb to haemonchosis, the greater part of the infection is thrown off in three to four months, and subsequently recovered sheep are found to have a considerable degree of resistance to reinfection.

The effects of the parasite are attributed chiefly to the loss of blood, and there is no evidence that harmful toxins are secreted. Loss of blood may cause the red cells to fall as low as 2 million per cc., and sudden deaths from extreme loss of blood are not infrequent.

Ostertagia causes a severe form of stomach-worm disease, and apparently it is increasing in prevalence and distribution in the United States. It has been identified in New York. Shaw[8] reports that it is common in sheep and goats in Oregon where *Haemonchus* is not found, and Welch[9] of Montana writes, "Of the stomach worms, *Ostertagia circumcincta* is by far more prevalent and troublesome than *Haemonchus contortus;* the latter does not constitute a serious problem for the western sheepman." *Ostertagia circumcincta* is the species commonly found in the abomasum in sheep, while *Ostertagia ostertagi* is the common cause of stomach-worm disease in cattle. These parasites are less widely distributed than *Haemonchus*. But they survive low temperatures, the eggs are said to develop at temperatures of 41° F., and apparently the larvae are extremely resistant to drying. Their habitat is

in the abomasum, where they may be so numerous as to cause undulating movements of the stomach contents which may be clearly visible when the semi-fluid is exposed for examination, or is poured into a shallow container. They are brown, hair-like worms varying in length from ¼ to ½ inch (7-10 mm.) and are often overlooked on postmortem examination because of their small size. The eggs are from 60-72 microns long by 42 wide. The larvae reach the infective stage in about

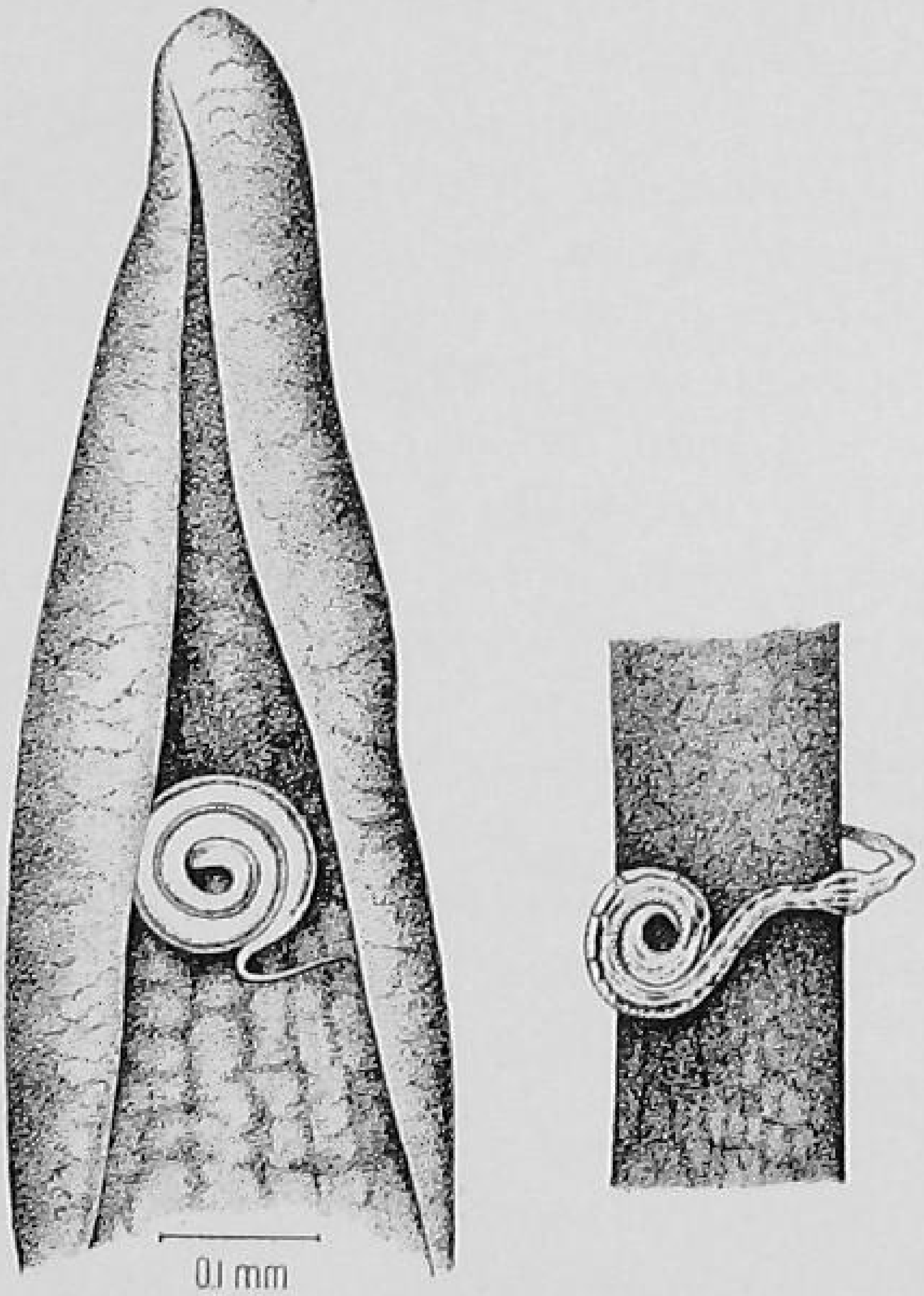

Fig. 24.—Larvae in infectious stage on blades of grass. Enlarged. Larva on grass blade at right shriveled but not killed by drying. The larva from which this drawing was made became active in a short time after being moistened with water. (Ransom, Cornell Veterinarian, 1920, 10, 66.)

a week. They burrow into the mucosa, causing the formation of small nodules, 1 to 2 mm. in diameter, within which they develop. They cause petechial hemorrhage, inflammation, and edema. The adults may form continuous masses embedded beneath a layer of mucus on the mucous membrane of the abomasum. They suck blood, and heavy infection may cause a high mortality in sheep and goats.

Trichostrongylus spp. is reported as the most wide-spread small strongyle affecting sheep and goats. In the United States its chief prevalence has been in the range sheep of the West from Montana to the

Coast, but the *Trichostrongylus* and other small strongyles are now widely prevalent in eastern sheep, where they may have been introduced in feeder lambs purchased in the West. There are several species: *Trichostrongylus axei*—(male 2.5-3.7 mm., female 3.2-4 mm. long) is found in the abomasum and anterior end of the duodenum of sheep, goats, horses, and cattle. *T. colubriformis—instabilis* (male 4.3-7.7 mm., female 5-8.6 mm. long) is found in the abomasum and duodenum of sheep and goats. *T. capricola* (male 3.5-5.8 mm., female 5.0-6.0 mm. long) is found in the abomasum and duodenum of sheep and goats. *T. vitrinus* (male 5.6-7.2 mm., female 6.8-8.1 mm.) is found in the duodenum and abomasum of sheep. In New York *T. axei* and *T. instabilis* have been recognized. These parasites are commonly termed the small intestinal worms but the habitat of *T. axei* is chiefly in the abomasum. Because of their small size (¼-⅓ inch) and reddish brown color, trichostrongyles are commonly overlooked on autopsy. They are credited with being chiefly pathogenic for the young (6 to 12 months), and *T. axei* is said to be severely pathogenic. The worms may be recognized by placing a scraping of the mucosa in a shallow glass dish of water and observing it against a dark background. The life history is similar to that of *Haemonchus*. Injury is caused by diffuse enteritis of the small intestine. In a number of instances this parasite has been the cause of severe losses in goat and sheep herds in New York, from which either *Haemonchus* has been largely eliminated by means of copper-sulfate treatment or was not originally present. Trichostrongylosis is readily transferable from sheep to cattle—Taylor.[10]

Concerning the pathogenesis of *Trichostrongylus*, the following statement appears in the Annual Report of the United States Bureau of Animal Industry for 1938: "Available evidence indicates that the pathogenicity of these worms is not associated with extensive hemorrhage in the intestinal tract since no anemia developed in the animals before death. Evidently the injuriousness of the worms was associated with certain marked changes in the chemical constituents of the sheep's blood, those changes being an increase in guanidine, blood sugar, and nonprotein nitrogen."

Morbid Anatomy.—The cadaver is greatly emaciated and an edematous swelling may be present beneath the lower jaw. On section, the peritoneal cavity contains a colorless serum. The internal organs are edematous and all of the tissues are pale. In the abomasum one finds large numbers of parasites. The gastric mucosa is diffusely reddened. In haemonchosis, hemorrhage into the abomasum causes a chocolate discoloration of the contents, and the mucosa often presents a reddish chocolate color. Unless the autopsy is held shortly after death the

worms may not be visible. Special attention should also be given to the small intestine in order to find the small Trichostrongyles embedded in the mucus.

Symptoms.—The disease usually occurs first among lambs late in the spring or in early summer, but it may occur at any season or age. The first signs are dullness, unthriftiness, pale mucous membranes, and a shaggy wool coat; though the victim may be found dead without recognition of previous symptoms. Sudden death in well-nourished sheep is attributed to loss of blood. In lambs the usual course is a week to ten days. In general the course in adults is over a period of weeks and months. Diarrhea is common, and in the later stages of the disease an edematous swelling may appear in the submaxillary region. In certain years, the mortality is high in both young and adults; in others, the loss is largely a failure to thrive and grow. Swampy pastures and wet seasons increase the severity. There is no age immunity.

Attempts have been made to describe the symptoms of stomach-worm disease according to the nature of infection, as haemonchosis, trichostrongylosis, etc., but in the majority of natural cases the infection is mixed and the symptoms are the result of a combined attack. For trichostrongylosis, emphasis has been placed on diarrhea with black feces, "black diarrhea," and absence of lesions sufficient to account for death. Perhaps death is caused by the chemical changes in the blood, as reported by the Federal Bureau of Animal Industry.

Diagnosis.—Autopsy of a recently killed animal is the most satisfactory method of diagnosis. Shortly after death the worms disintegrate and consequently are not found. To detect the smaller worms, take a scraping from the mucous membrane, shake in water, and examine in good light. A low-power microscope is still better, since the small worms are easily missed without magnification. *T. axei* is not readily visible to the naked eye. *Haemonchus* is readily detected and identified without magnification. The presence of many eggs in the feces indicates stomach-worm disease, but differentiation between the different species by examination of the eggs may be impossible. Furthermore, severe symptoms may be caused by heavy infection with larval worms—Mahew.[11] For an accurate identification of adult worms, it may be necessary to submit them to a laboratory (Zoological Division, Bureau of Animal Industry, Washington).

For various reasons, failure to make an accurate diagnosis in stomach-worm disease is rather frequent. One may fail to search for *Trichostrongylus* and other smaller parasites when they occur in association with *Haemonchus*, or, not finding *Haemonchus*, may discontinue the search. Postmortem disintegration may have removed all traces of

parasites. A negative conclusion can be reached only after microscopic examination of the stomach contents, preferably from a recently killed animal.

Treatment.—A solution of equal parts copper sulfate and 40 per cent nicotine sulfate—Black Leaf 40 (1.5 per cent of each in water) is regarded as the most effective known combination for the expulsion of large and small strongyles (*Haemonchus*, *Trichostrongylus* spp., and *Ostertagia*). Dissolve 2 ounces (60 Gm.) of copper sulfate in a gallon (8 liters) of water and add 2 ounces (60 cc.) of Black Leaf 40. The dosage is as follows:

Adults	90 cc.	(3 ounces)
Yearlings	60 cc.	(2 ounces)
6-month-old lambs	40 cc.	(1⅓ ounces)
3-month-old lambs	20 cc.	(⅔ ounce)

Or:

80-100 pounds	3 ounces	(90 cc.)
60 pounds	2 ounces	(60 cc.)
30 pounds	1 ounce	(30 cc.)

Medication should not be preceded by starvation, and the animals may be turned loose at pasture directly after medication. In heavily infected areas this treatment may be repeated every three weeks. It is regarded as the most effective combination against small intestinal worms and it is also a specific for tapeworms. Gordon and Ross[12] concluded that "even though exposed to heavy infection with *Trichostrongylus* spp., sheep may be protected by routine treatment at intervals of three weeks with copper sulfate and commercial nicotine sulfate solutions." Repeated failure of individual sheep to respond to treatment with copper sulfate-nicotine sulfate mixture administered against *H. contortus* has been explained by Gordon and Whitten[13] as due to the failure of the esophageal groove to close in certain individuals; such cases can be treated with carbon tetrachloride.

For years copper sulfate has been a widely used and effective vermifuge against *Haemonchus contortus* of sheep, but it failed against the small strongyles (*Trichostrongylus*, *Ostertagia*). In 1934 Ross[14] made the important observation that a solution of copper sulfate given orally induces reflex closure of the esophageal groove, causing the fluid to pass directly into the abomasum. The passage of fluids into the abomasum was caused by the presence of copper sulfate and not by starvation, as previously believed. This suggested the possibility of copper sulfate as a vehicle in which other drugs might be administered directly into the abomasum. And subsequent experiments proved that

when the administration of copper sulfate solution is followed immediately by another solution, it also passes into the abomasum. Effective action follows the use of 2.5 cc. of a 10 per cent solution given orally, or the mouth may be wiped with a swab soaked in the solution. The reflex action lasts for about 15 seconds. Discovery of this specific action has resulted in the development of more effective methods of combatting parasites of the intestines. A 10 per cent copper sulfate and 10 per cent black leaf 40 (10 per cent cunic) solution is recommended by Whitlock[15] as economic and efficient in the control of gastrointestinal helminthiasis in sheep; an apparatus for treating 200 sheep an hour is described.

Tetrachlorethylene has been reported by Shaw[8] as an effective treatment of stomach-worm disease of sheep in Oregon; it is administered in capsules of 5 cc. each. The most effective use of tetrachlorethylene is obtained when it is combined with equal parts liquid petrolatum and given directly after a small dose of copper sulfate. The recommended dose of the mixture is 10 cc. for adults and 5 cc. for lambs over 6 months old. Mönnig[16] prescribes 7.5 cc. for sheep over 9 months old and 5 cc. for lambs, mixed with an equal quantity of liquid petrolatum, and given immediately after 2.5 cc. of 10 per cent copper sulfate solution.

In the medication of sheep for stomach-worm disease the animals should not be driven directly before or directly after treatment; they should be handled quietly, and unthrifty individuals should receive less than the prescribed dose. The medication should be repeated at least once after an interval of ten to fourteen days.

Phenothiazine is widely reported as efficient against the chief infestations in stomach-worm disease in sheep. A review by Davey and Innes[17] of the reports on phenothiazine includes this statement: "Most workers affirm that phenothiazine is virtually 100 per cent efficient in removing *Haemonchus contortus*. It is less efficient, but still very good, in removing *Trichostrongylus axei*, *Ostertagia circumcincta*, and *O. trifurcarta*. All of these species, it will be noted, are from the abomasum. It will remove the immature stages of *H. contortus*, which is most important because it is then unnecessary to repeat treatment at short intervals."

The following recommendations for the use of phenothiazine have ben made by Thorp[18] and associates: "Treat all the sheep in the flock in the late fall and winter with the phenothiazine drench. Repeat this treatment in the spring 1 to 2 weeks after all the ewes have lambed. Then, place the entire flock on the 1:9 phenothiazine and salt mixture. Drench the lambs in the flock with phenothiazine when they are 3 to 4 months of age. If it is a wet, warm season and the lambs show any

evidence of parasitism, it may be necessary in some cases to drench them again. Under some conditions, particularly after using the fall and spring drench for several seasons, it may be necessary to drench the ewes in the fall if they were drenched in the spring, providing the 1:9 salt mixture has been before them at all times. All of the young stock kept over the winter should be drenched in the fall." The 1:9 phenothiazine and salt mixture is prepared by mixing 1 lb. of phenothiazine powder with 9 lb. granular salt. For lambs weighing up to 60 lb., the dosage of phenothiazine is 15 Gm. It is well tolerated by pregnant

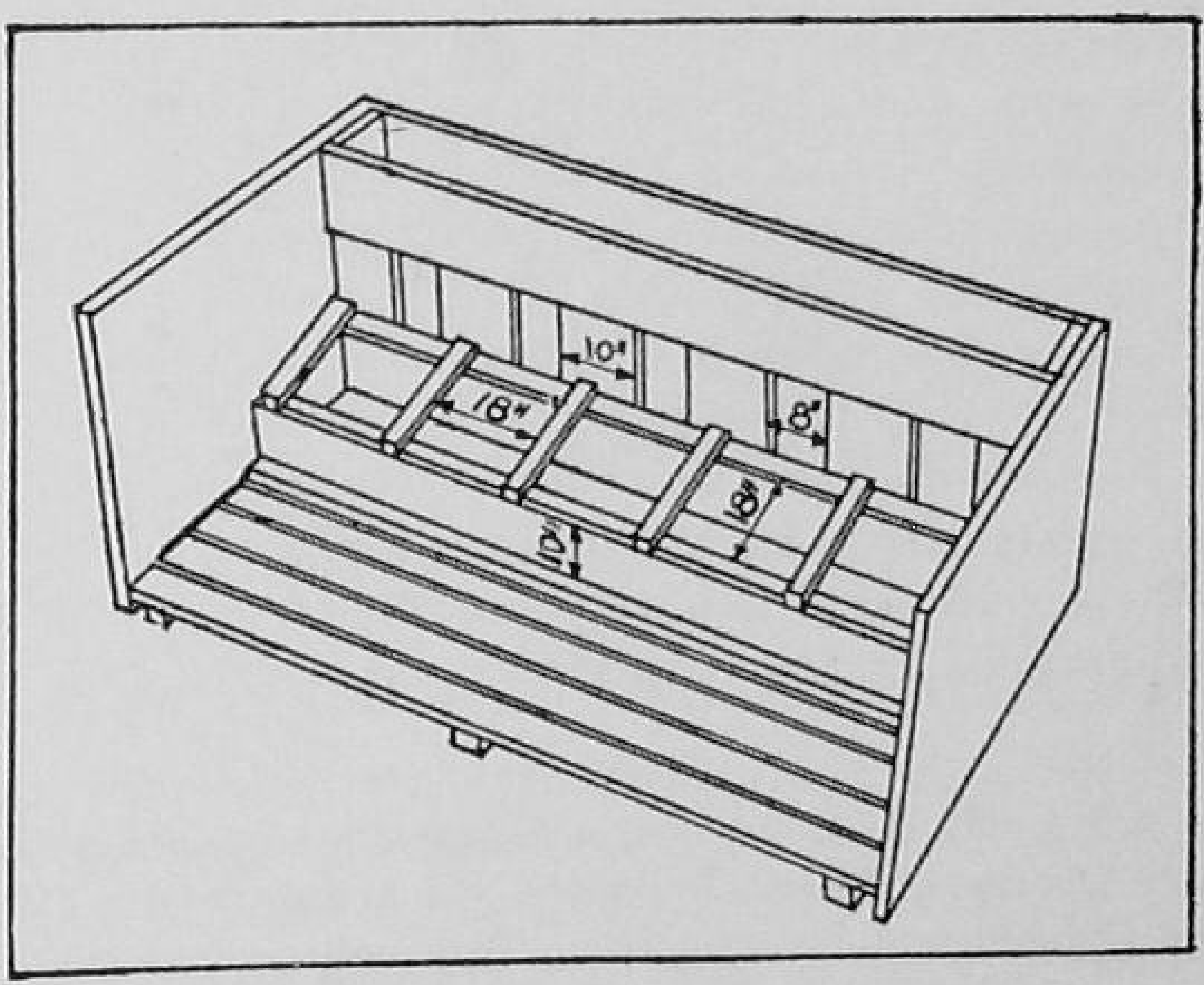

Fig. 25.—Sanitary feeding rack used in control of parasites. (Turner.)

ewes, and can be administered at a dosage of 25 Gm. in the feed without risk to either the ewes or their lambs. Phenothiazine may also be fed in soybean-meal pellets—Thorning et al.[19] In badly diseased individuals the dose should be reduced one-half. In numerous recent articles on the use of phenothiazine, the majority recommended salt licks combined with occasional drenching; others report success with salt licks alone, while a Texas report indicates the need for pasture rotation in addition to the use of anthelmintics. Seghetti and Marsh[20] report from Montana that "ten per cent of phenothiazine in salt at the rate of 1.5 Gm. of phenothiazine per day for each ewe and her lamb will not only prevent clinical nematode parasitism in the lambs, but will reduce worm numbers to a minimum in the ewes . . . winter treatment with phenothiazine of ewes not showing clinical parasitism is not profitable on a range where sheep will take salt in the summer."

A suspension suitable for drenching is prepared by mixing 500 Gm. phenothiazine with an equal amount of molasses and enough warm water to make a total volume of 2000 cc. The dose for mature sheep is 120 cc., and for lambs weighing between 50 and 70 pounds 60 cc. Administer with a dose syringe and stir frequently to maintain suspension. A rapid method of drenching sheep with an esophageal tube attached to a syringe at a possible rate of 200 sheep an hour has been described by Whitlock.[15]

Prophylaxis.—The following recommendations are made with respect to the prevention of parasitism: feed well, give a vermifuge at regular intervals, avoid overstocking of the pasture, separate young from adults early, and avoid damp places and water holes favorable to the development of parasites. Fecal contamination may be restricted by raised troughs and hayracks. A specially constructed feeding rack has been designed by Turner[21] (Fig. 25). The rack is set a foot above the floor and is constructed of slats wide enough apart to admit a sheep's head. About 18 inches back of the rack a 10-inch board is set on edge. To reach the hay sheep must step over this board with the front feet, and to keep them from getting in with all four feet, strips are nailed every 18 inches from the board to the rack. This prevents them from dragging the hay out and tramping it under foot.

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STOMACH-WORM DISEASE IN CATTLE

Etiology.—*Ostertagia ostertagi* has been occasionally mentioned as a cause of stomach-worm disease of cattle in the United States. Apparently the first record of this disease in this country was by Stiles.[1] In 1900, in an account of verminous disease in cattle, sheep, and goats in Texas, he described the symptoms, autopsy findings, and parasites. In May, 1920, Muldoon and Frick[2] described an outbreak in 84 breeder steers bought in the Kansas City yards. A more complete description of this outbreak was made by Ackert and Muldoon[3] in November, 1920. In 1927 Barger[4] reported the deaths of 11 yearlings from this disease in the San Joaquin Valley in California. In the California Station Report for 1927 it is stated that Barger's experience with this condition is probably the first to be reported in California and it is the third to be reported in the United States. In 1928 it was observed in Illinois by Hawes.[5] In the summer of 1931 it was the cause of several deaths among cattle in Penn Yan, New York; these animals had recently been shipped from the West. In the cattle observed by Hawes, *H. contortus* was also present. Although this disease has been reported from widely separated sections of the United States, it does not appear to very frequent. It is reported from European and other countries as an occasional enzootic in young cattle. In 1905 Klein[6] wrote a bulletin on verminous

gastritis of calves caused by *Haemonchus contortus*. He stated that, "in this country it affects calves and young cattle, although perhaps not as commonly as sheep. In the United States, the records report the finding of the worm only in Maryland, Texas, and District of Columbia cattle, but it is believed the disease is much more widespread. In South Carolina, the disease is not a recent importation. . . . One correspondent, who reported his cattle affected last fall, said he believed his cattle had suffered from the same disease ten years ago." According to Baker[7] the most common nematode parasites in calves in New York State are

Fig. 26.—Photograph of a calf which shows the condition produced by heavy infection with *Ostertagia*. Cornell Vet., 1937, **27**, 381. (Courtesy of D. W. Baker.)

Ostertagia (abomasum) and *Nematodirus* (small intestine). Other parasites that may be associated with *Ostertagia* in stomach-worm disease of calves are *Cooperia oncophora* (small intestine) and *Trichostrongylus*. Special importance attaches to the habitat and activity of this group of parasites. It has been reported by Taylor[8] that infective larvae of *Ostertagia* and *Nematodirus* can regain the surface of the pasture after being turned under by the plow and retain their infectivity for a year, that mass development of infective larvae may take place in as short a time as six days under field conditions, and that from soil infection ostertagiasis may develop in as short a time as four weeks.

Ostertagia ostertagi is the most frequent cause of stomach-worm disease in calves. Stiles[1] writes that the parasite was found in every

calf, steer, and cow examined on his second trip to Texas, and Marek reports that in the Berlin abattoir, Ostertag found it in **90** per cent of the slaughtered cattle. Infection is favored by overstocking of pastures, fertilization of pastures with manure, drinking contaminated surface water from water holes and moist places, and swampy pastures.

Morbid Anatomy.—Stiles emphasizes the small size of *Ostertagia*,

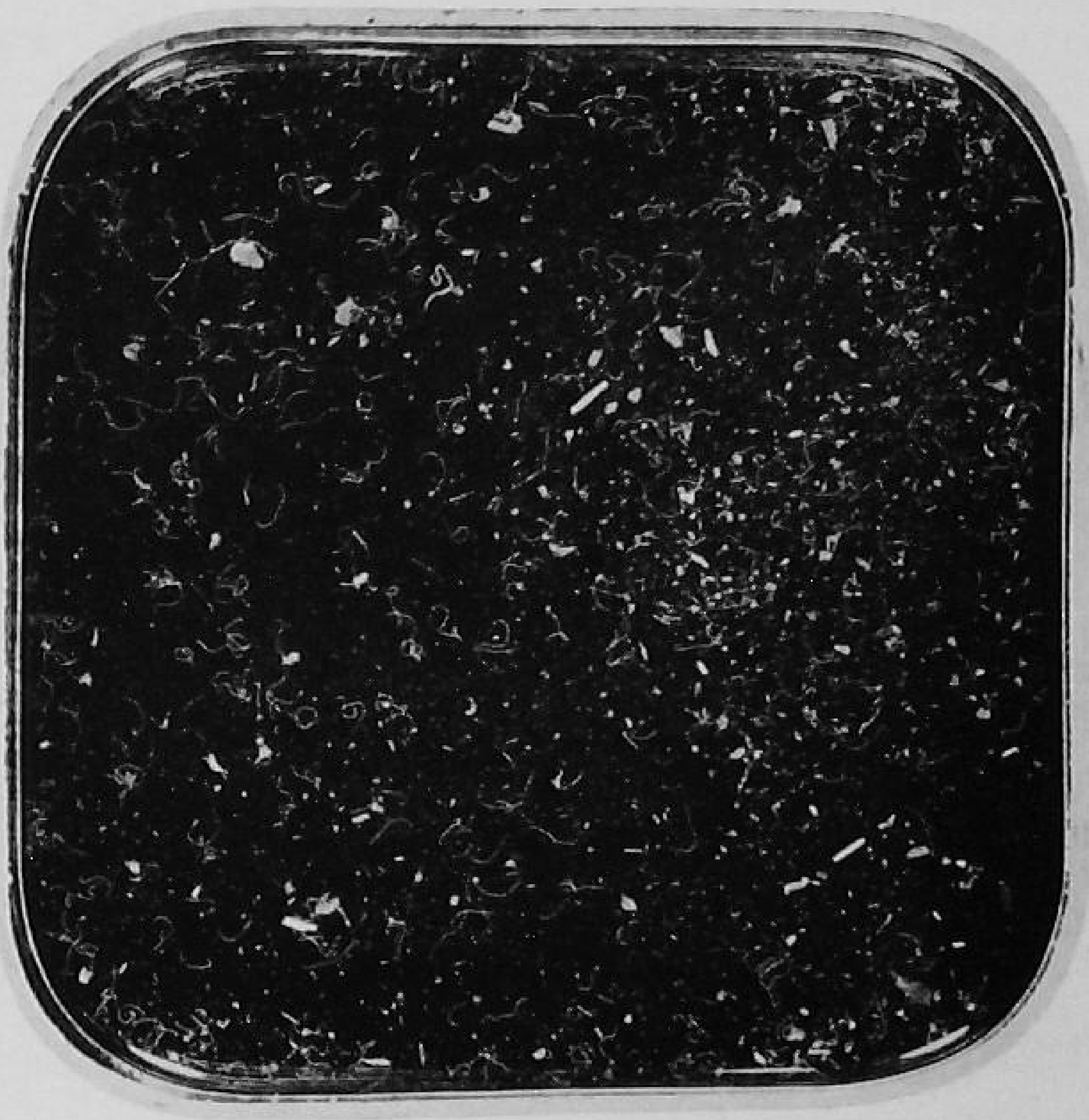

Fig. 27.—Photograph of a small sample of stomach contents which had been poured into a square petri dish. The sample has been diluted in saline solution in order to spread out the worms which are reproduced in actual size. Cornell Vet., 1937, **27**, 381. (Courtesy of D. W. Baker.)

about the thickness of a fine hair and less than one-half inch in length, and the chances against its being discovered unless one's attention is especially called to it. *Ostertagia* may be free in the ingesta, and it may also be encysted in the wall of the stomach. Numerous small nodules with minute openings are on the mucosa, and worms may be pressed out by scraping with a knife. In many of the cases in Texas the stomach was literally studded with these parasites. The stomach wall was sometimes found to be one-half to one and one-half inches thick;

it was edematous and pressure caused a large quantity of fluid to flow from it. The cadavers were anemic and the hind parts were smeared with feces. In the cases reported by Barger[4] the large and small intestines presented changes suggestive of Johne's disease. An autopsy is best performed on a recently slaughtered animal when the worms are still alive and active. Because of the small size of *Ostertagia* they may not be visible in the exposed stomach contents, even when present in large numbers. Their presence may readily be detected, however, by the

Fig. 28.—Photomicrograph of a portion of the field visible under low power magnification of a fecal smear. The sample taken from the feces of an infected calf shows the ova of the worms on a background of fecal debris, *a* is a typical egg from the stomach-worm *Ostertagia ostertagi*, *b* is an air bubble, and *c* is the embryonated egg of a grain mite. Cornell Vet., 1937, **27**, 381. (Courtesy of D. W. Baker.)

constant undulating movement of the fluid in the abomasum. When a little of this material is diluted with saline solution and poured into a petri dish the small worms are visible (Fig. 27).

Symptoms.—The majority of cases that have been described in this country have developed in the fall or winter, chiefly in yearlings at pasture, but the disease may appear at any time during the pasture season. The principal symptoms are loss in condition, pale mucosae,

temperature from normal to 104°, a profuse watery diarrhea, and weakness. Bloody diarrhea has been observed. In a few days edematous swellings may appear in the submaxillary region. The appetite is usually good. In the cases described by Muldoon,[2] death occurred in about a week after the appearance of profuse diarrhea. In Klein's cases, the course was two to three months after showing the first symptoms. Because of the changes in the wall of the stomach induced by *Ostertagia*, disease caused by this parasite is often fatal.

Treatment.—Tetrachlorethylene and liquid petrolatum equal parts (15-20 cc. per 100 lbs. body weight) administered directly after giving 2.5 cc. of 10 per cent copper sulfate solution, or swabbing the mouth with the copper sulfate solution, has proved to be effective in the removal of *Ostertagia*.[9] The treatment may be repeated once or twice at intervals of ten to fourteen days. Do not starve and do not drive animals before or after treatment.

Phenothiazine is reported by Davey[10] and Swanson,[11] to have an efficiency in calves comparable to that in sheep. It has a low toxicity for calves and the dose is 20 Gm. per 100 pounds (45.4 kg.) up to a maximum of 60 Gm. per calf, administered after a fast of from 18 to 24 hours. Small weak calves should not receive more than 20 Gm. It may also be given in the 1:9 phenothiazine and salt mixture as directed for sheep (p. 208). In calves phenothiazine is highly efficient against *H. contortus*, *Trichostrongylus axei*, and *O. ostertagi;* to get the *Haemonchus* it should be repeated in not less than 21 days.

In advanced cases of trichostrongylosis in calves, dried yeast has been reported by Baker[12] to be a useful adjunct; one-half pound was given twice daily.

Phenothiazine given in a dosage of 60 Gm. to unweaned beef calves 5 to 7 months old reduced internal parasites and promoted growth; there was no difference in the number of small intestinal worms (cooperids), but only one-third as many stomach worms and hookworms were found in the treated calves as in those untreated, 2 months after treatment.[13]

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ANIMAL PARASITES IN THE SMALL INTESTINE

ASCARIASIS IN SWINE

Definition.—Ascariasis in swine is an affection of young pigs caused by migration of larvae through the liver and lungs, and the presence of adults in the small intestines. Death or injury to the pig is caused by lesions in the lungs or liver, or by toxic substances secreted by the adult. The specific cause is *Ascaris lumbricoides* (*Suum*). Wherever the raising of swine is an important industry in this country, ascariasis is a serious parasitic disease. Because of its great economic importance, federal and local governments have instructed and demonstrated extensively in methods of prevention, and the "McLean County system" (Raffensperger)[1] is widely known.

Life History.—The female is 6 to 12 inches in length, the male 4 to 6 inches. It is cylindrical in form, pointed at both ends, and colored yellowish-brown or slightly red. The eggs are oval, 60 to 75 microns long, by 40 to 58 wide; the shell is mammilated and the color is yellow. The habitat of the mature parasite is in the small intestine, but it may migrate to the bile ducts, the pancreatic duct, or the stomach.

Ransom and Foster[2] were able to infect swine artificially by feeding ascarid eggs containing motile vermiform embryos to pigs two weeks old. They found the life history to be direct without intermediate host. It is given by Ransom[3] as follows: "The eggs as they are deposited by the worms in the intestine and as they are passed out in the feces of the infected animal, are in early stages of segmentation. At the temperature of the body the contained embryos are unable to complete their development to the infectious stage, but at the lower temperature of the outside world in the presence of oxygen and moisture, the embryos finally reach the infectious stage after which no further development occurs until the eggs are swallowed. The eggs may become infectious as early as ten days after they have passed in the feces but usually a much longer time is required. They are highly resistant to unfavorable conditions such as cold or dryness, and the embryos protected by the shell of the egg may retain their vitality for long periods of time. Embryos kept for five years have been found to be still alive (Devaine).[3] From these facts it is evident that soil that has been contaminated by the feces of infested pigs will retain infection for a long time; also that the amount of infection in places occupied by pigs will tend to increase so that ultimately the soil will become heavily laden with ascarid eggs.

"When sufficiently incubated eggs are swallowed by a pig, they hatch in the small intestine, but the young worms do not immediately settle down. Some of them may be carried out of the body in the feces and these soon perish. Others leave the lumen of the intestine and migrate to the liver, lungs, and various other organs, most of them going apparently to the liver, and then to the lungs, probably aided in their migration by the circulation. Those that do not succeed in reaching the lungs, according to the evidence at present available, undergo some

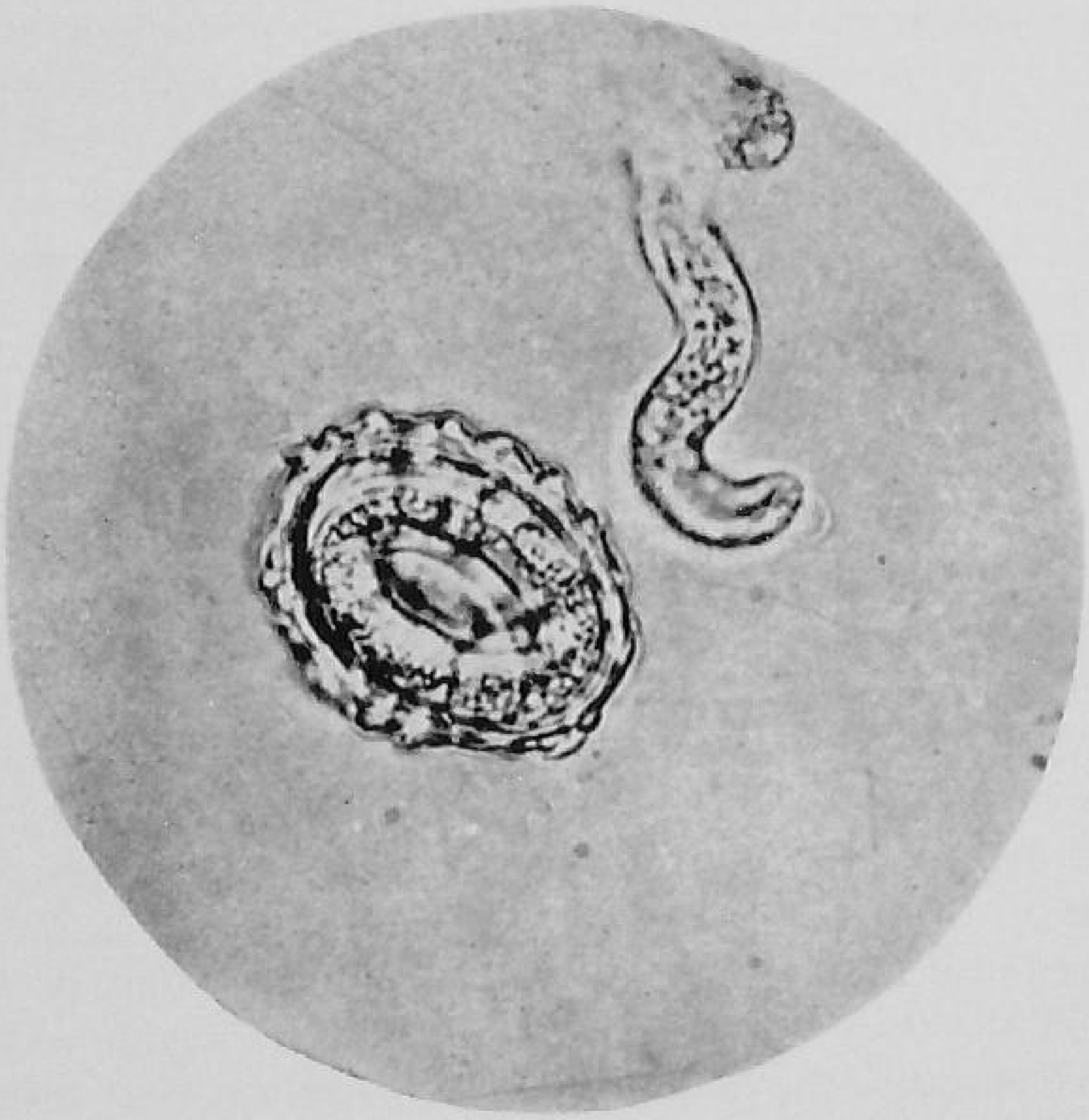

Fig. 29.—Egg of *Ascaris lumbricoides* in the infective stage showing embryo coiled within the shell. x 400. (Benbrook, Veterinary Practitioner's Bull., Ames, Iowa, Iowa State College, 1925, **24**, No. 10.)

development but soon die and degenerate. Those that reach the lungs during their migrations grow and develop to a stage considerably more advanced than the newly hatched larvae, becoming five to ten times the original length, 1.5 to 2.5 mm. as compared with 0.25 to 0.3 mm. In cases of heavy infection they may be found in large numbers in the lungs a week to ten days after the eggs have been swallowed. . . . From the lungs the young worms crawl up the trachea and down the esophagus and pass through the stomach and into the small intestines, where

they may be found as early as six days after infection, but in considerable numbers not until ten days have elapsed after the eggs were swallowed. They establish themselves in the small intestine and complete their development to maturity. From the time the eggs are swallowed about two and one-half months are required for development to the adult stage."

Fig. 30.—Section of mouse lung showing Ascaris larva. (Ransom, Cornell Veterinarian, April 1920.)

Morbid Anatomy.—Pathological changes in the liver that result from migration of the larvae are first congestion and hemorrhage. Following migration the liver shows numerous whitish indurated areas about ¼ inch in diameter. The changes in the lungs are edema, petechial hemorrhage, and lobar or lobular pneumonia. The pig is undersized and poor. The skin may be jaundiced. In the bronchi and intestines are numerous larvae. If there has been time for their growth, mature ascarids are found in the intestines.

Symptoms.—Susceptibility is chiefly among little pigs, 2 to 8 weeks of age; older animals and adults are relatively immune to new attacks.

It is generally believed that a few parasites cause no disturbance, yet Kernkamp[1] reports that in clinical cases the number of worms present, visible to the unaided eye, was often as few as 20. In dirty pens suckling pigs often acquire a heavy infestation that may lead to death in a week to ten days. The chief symptoms are unthriftiness, anorexia, vomiting, cough, "thumps," and dyspnea. The larvae are a frequent cause of disease of the lungs in pigs. Recovered pigs are permanently

Fig. 31.—Stunting caused by Ascaris infection. Pig in middle passed through an attack of pulmonary ascariasis. All pigs under same conditions as to age, feed, and care. (Ransom, Cornell Veterinarian, April, 1920.)

stunted. Occlusion of the bile ducts causes jaundice and convulsions, often death. A poor condition and an icteric skin in an otherwise normal pig is an indication of ascariasis, and such individuals may succumb to a simultaneous vaccination against hog cholera. Numerous larvae may be found in the feces and the bronchial mucus of an affected pig.

Adult parasites in the intestines secrete an irritating substance that may cause severe catarrh. When this substance is brought in contact with the skin of certain individuals, through handling the parasites, an intense itching results; the same effect may be experienced from a rectal examination of a heavily infected horse. The blood undergoes hemolysis and eosinophilia may develop.

Treatment.—Approximately three-fourths of the worms are expelled from the intestine by a single dose of oil of chenopodium: 5 cc. per 100 pounds in 2 ounces (60 cc.) of castor oil or mineral oil. Withhold food for twelve to twenty-four hours before treatment. For heavily infected swine reduce the dose one-half and repeat after an interval of twelve hours. This may be repeated in a week or ten days in order to remove any that may have escaped the first treatment. It may be given in the mouth with a dose syringe or placed directly in the stomach through a tube. The castor oil should never be omitted. While this vermicide is highly effective in the removal of adult worms from the intestine, it does not repair the injury that has resulted from invasion of the lungs. During anthelmintic treatment, pigs should be kept in a place that can be cleaned and disinfected after the expulsion of the worms.

According to Swanson[5] and associates phenothiazine administered to pigs as an anthelmintic seems to be as effective for the removal of mature ascarids as oil of chenopodium, except in cases where only a few worms are present. The dosage for pigs of different weights is: under 25 lbs., 5 grams; 25 to 50 lbs., 8 grams; 50 to 90 lbs., 12 grams; 90 to 175 lbs., 20 grams. It may be administered in capusules or in the feed, and it has a low toxicity for swine.

In an extensive comparison of the relative value of oil of chenopodium, phenothiazine, and sodium fluoride, Enzie[6] and associates report that oil of chenopodium appears to remove about three-fourths of the ascarids from pigs, and that phenothiazine is considerably less effective; "In limited trials, sodium fluoride demonstrated a significantly superior ascaricidal action in pigs, was as well tolerated as either of the above-mentioned drugs, and was easy to administer. Much more extensive testing will be necessary, however, before the safety and anthelmintic value of sodium fluoride can be properly evaluated." It is administered as 1 per cent of the feed for one or two days. An additional favorable report on trials with sodium fluoride has been made by Allen.[7]

In a U.S. BAI Report for 1948,[8] it is stated that 0.75 per cent of sodium fluoride appears to achieve essential maximum efficacy and safety with no detectable loss of natural safeguards; this concentration produced slight congestion of the kidneys in a few pigs and reduced the feed intake from 31.3 per cent of the normal in some pigs and 43.2 per cent in others.

Prophylaxis.—Prevention is the most effective method of combating ascariasis in pigs. This should begin with the treatment of the sow about to farrow. The McLean County system is as follows:

"1. Clean the farrowing quarters thoroughly and then scrub with boiling water, adding 1 pound of lye to 30 gallons of water.

"2. Brush loose litter and mud from sides of sows; then wash the udder thoroughly with warm water and soap, and then place the sow in the clean farrowing pen. This is done three or four days before farrowing.

"3. Confine the sow and pigs to the farrowing pens until they are moved to clean pasture, and haul—do not drive—them to the pasture. Water and feed must be provided in the clean pasture, as the young pigs must not, under any circumstances, be permitted to go back to the permanent hog lot for feed or water until 4 months old. It is advisable to confine them to the pasture until time to turn them into the cornfield."

Routine treatment of the sow before farrowing for the expulsion of ascarids is not practised. Sows are less heavily infested than the young pigs. According to Raffensperger, "experiments indicate that at the time of the March and April farrows it will probably take at least eight weeks, under weather conditions prevailing in Illinois, for the eggs passed in the manure of the sows to become infective."

Eliminate hog wallows, and drain yards and pens. Remove manure often to fields not occupied by swine. Badly infected lots should be plowed and kept free of hogs for a time.

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THORN-HEADED WORMS IN SWINE

Macracanthorhynchus hirudiniaceus (Echinorhynchus gigas), about the size of an ascarid, occurs in the small intestine of swine and especially in the jejunum. It is reported by Mönning to have a wide distribution, but in this country it is chiefly a parasite of the South and other areas where hogs pastured in wood-lots are infected by eating beetles and grubs. Damage results from irritation of the mucosa and perforation of the intestinal wall. Diagnosis can be made by finding the eggs in the feces. No effective remedy has been reported.

ASCARIASIS IN EQUINES

Ascaris equorum (megalocephala.)—This species is found only in equines. Its habitat is the small intestines, and occasionally it migrates to other organs. The female is 6 to 12 inches long. The eggs are round and 90-100 microns in diameter. According to Hobmaier, and Hadwen[1] the life history is analogous to that of *A. lumbricoides*. Many horses harbor a few of the mature parasites when they do no harm. Colts suffer severely from heavy infections, and older animals may likewise be affected. Infection occurs through contaminated food and water. This most readily happens when roughage is eaten off the floor where it has been exposed to fecal contamination. Young foals may lick the walls and floor, nibble at bedding or feces, and even develop the habit of eating filth. Foals that run on permanent pasture are always infected from this source. Confinement in dirty stables favors infection. The mature parasite causes mechanical injury and forms harmful products that affect the general condition, the blood and the nervous system, as in swine. Migrating larvae injure the liver and lungs. A combined infection with ascarids, bots, and strongyles is frequent.

Dimock[2] has reported from Kentucky that all horses that have died following an attack of sickness have shown on autopsy parasites and tissue changes resulting from the presence and action of parasites that cannot help but have been a factor in bringing about sickness and death; he believes it would be worth while to adopt a system of parasite control on every horse farm.

Morbid Anatomy.—In the young foals described by Hadwen,[3] the lungs were spotted, edematous, and slightly hepatized in spots. The liver was badly spotted and degenerated. The small intestine of one was hard, thick, and contained much mucus. Ascaris larvae, varying in length from 2 mm. to 3 cm., were found in the duodenum, and they were also numerous in the air passages. In one badly stunted 2-year-old colt autopsied in our ambulatory clinic the small intestine was filled

for 20 feet with ascarids; the walls were thick and yellow and the mucosa was congested, the mesenteric lymph glands swollen, and the abdominal cavity contained clear serum. Dimock[2] reports having seen two or three cases of rupture of the intestine by ascarids.

Symptoms.—In *young foals* migration of the embryos through the lungs causes bronchitis and pneumonia and may prove fatal. Recovered animals continue to cough and discharge from the nose for some time.

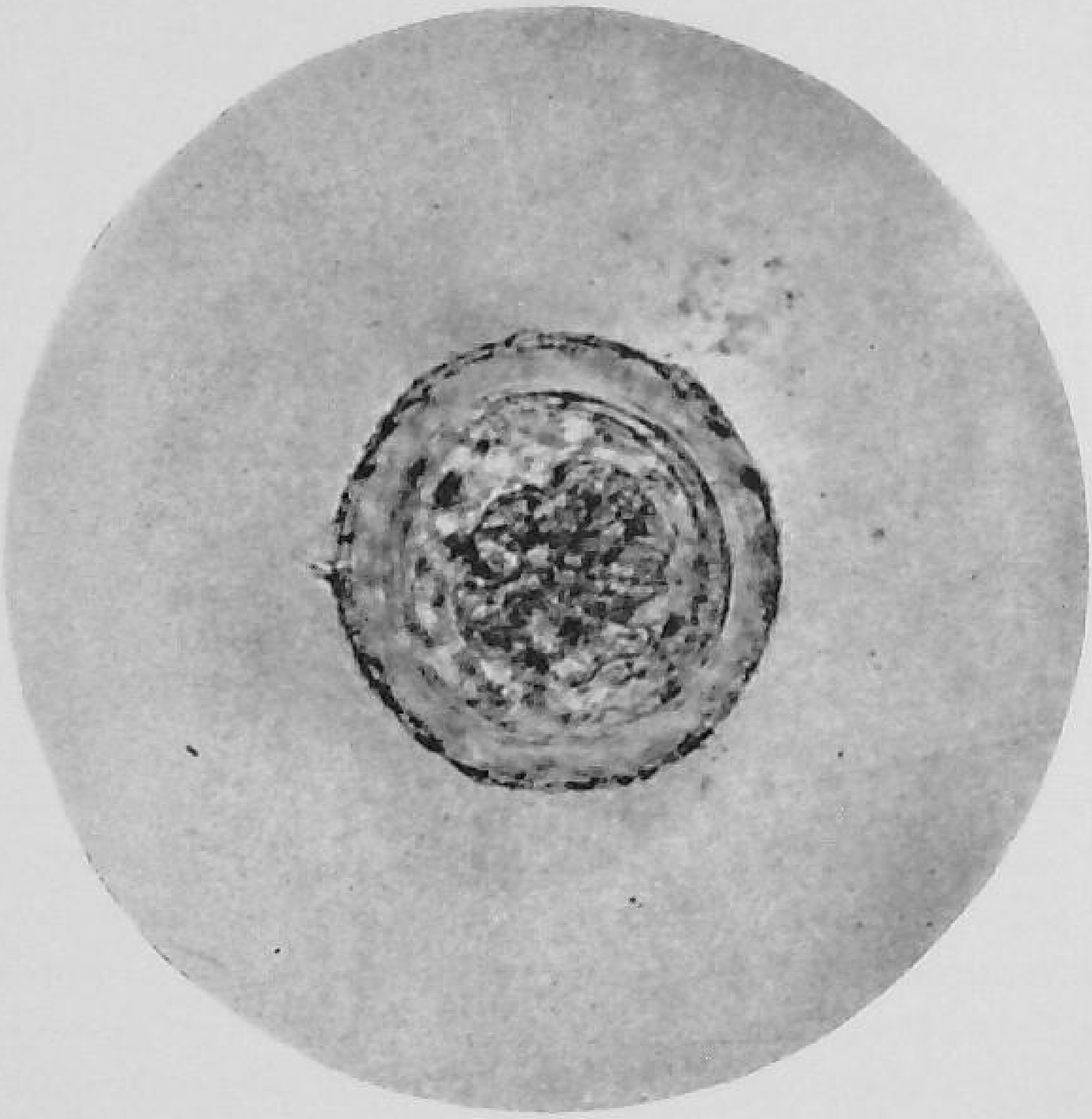

Fig. 32.—Egg of *Ascaris equi* x 400. (Benbrook, Veterinary Practitioner's Bull., 1929, **27**, No. 49.)

In addition to dullness and poor condition, the pulse, respiration, and temperature may be above normal. Heavily infested *yearlings and two-year-olds* are half-grown, emaciated, weak, and without appetite. In extreme involvement there may be nervousness, twitching of the muscles, great thirst, icterus, a weak rapid pulse, and an increased heart impulse. While this disease chiefly attacks the young, *adults and old horses* are often affected. Such animals present a history of gradual loss of condition in spite of good appetite and abundant food. They are poor and dull, and tire easily. The hair is long and rough and the skin dry and dirty. The mucous membranes are usually congested. The abdomen tends to be small and pendulous and the peristalsis to be

decreased. The feces are dry and brown—"burned"—and covered with mucus. In young animals constipation may alternate with diarrhea. A rectal examination may cause itching and redness of the skin of the examiner. Sometimes a few ascarids are passed in the feces. On microscopic examination of the feces, eggs are often found; but only when they are numerous can a positive diagnosis be made. In suspected cases a vermifuge will bring evidence of the number of adult parasites.

Diagnosis.—This is made on the history of gradual loss in condition, the evidence of anemia and debility, and the presence of numerous eggs in the feces. In foals the lesions in the lungs and liver, combined with the presence of larvae in the duodenum and bronchi, afford a means of diagnosis when the parasites have not approached maturity in the small intestine.

Treatment.—Carbon disulfide 6 drams (25 Gm.) has been found by Hall[4] and associates to be almost 100 per cent effective in the removal of ascarids. Food is withheld for 18 to 24 hours preceding medication. The vermifuge is given through a stomach tube or in a capsule, and 4 hours later the horse is fed. The dosage according to age is as follows: 3 to 5 months, 10 cc.; 5 to 8 months, 15 cc.; 12 to 18 months, 20 cc.; 2 years, 25 cc.; 3 years and over, 30 cc. There may be constipation in young horses two to four days after administration, and this may be relieved by giving 1000 cc. of liquid petrolatum. The administration of carbon disulfide should not be combined with a laxative.

Carbon tetrachloride 6 to 12 drams (25-50 cc.) per 1000 lbs. or more is also effective for the removal of ascarids. This should be followed by a saline purgative (sodium or magnesium sulfate 1 lb. dissolved in water) to reduce the toxic effect, and if the animal is poor include calcium gluconate. Give a bran mash at night of the first day, fast the second day, administer carbon tetrachloride on the morning of the third day, and feed at noon. Carbon tetrachloride may be given to pregnant mares. In the use of carbon disulfide or carbon tetrachloride water should be provided before treatment, but not for several hours after treatment.

In our experience, tartar emetic administered according to Grimme's[5] method has proved satisfactory. Withhold water at night; dissolve 4 to 5 drams (15-20 Gm.) in a pail of water; give one third at 6 A.M., one third at 7 A.M., and one third at 8 A.M. Colts six months to one year old receive 5 to 10 grams, suckling foals 2 grams. Fowler's solution of arsenic 4 drams (25 cc.) given three times a day may be followed by the expulsion of many worms.

Prophylaxis.—Wash and disinfect the foaling stall previous to parturition; turn mare and foal in a paddock or pasture not previously

occupied by equines for at least a year; clean stables frequently to avoid fecal contamination; and lime the floors. Bardwell has observed that removal of manure once a week from the pastures of the studs in Kentucky will reduce infection in foals.

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ASCARIASIS IN CALVES AND LAMBS

Ascariasis in calves and lambs is rare in the United States. In Southern Europe, calves frequently harbor ascarids. By some the species is regarded as identical with that affecting children and pigs—*A. lumbricoides*, although it carries a different name: *A. vitulorum*, calves; *A. ovis*, lambs. According to Gasteiger the worms are removed by tartar emetic (3 to 5 Gm. in 125 cc. water) given in doses of 15 cc. every 4 hours until the worms appear.

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TAENIASIS IN SHEEP

(Tapeworm)

Taenia fimbriata (Fringed Tapeworm; *Thysanoma actinoides*).—This parasite is common in the United States west of the Mississippi, but it is not known to occur in flocks east of this river. In 1890, Curtice[1] reported it as a native of North and South America, as the most common parasite of our Plains, and one that causes great loss. In Colorado he found that 80 to 95 per cent of the sheep in the flocks were affected and that as many as 100 parasites might be found in a single sheep. Hall[2] writes that it seems to be losing ground with the breaking up of the big western sheep ranges, and Welch[3] states that "The fringed tapeworm was considered a very important parasite of lambs a few years ago, but we do not consider it so now." Heavy infection, however, is a cause of considerable loss from condemnation of livers in packing houses.

Life History.—The habitat of the mature form is the small intestine,

and young parasites grow in the bile duct. Its usual length is about 6 inches though it may reach a foot. The posterior border of each segment has a fringe or projection—fringed tapeworm. Segments containing eggs pass out of the sheep in the feces, but the further development of the parasite is unknown; an intermediate host is suspected. The smallest forms of the parasite appear in lambs soon after the second month of age, but it requires at least six months, possibly ten, to attain adult size. Because of the slow growth it affects lambs of a more advanced age than those infected with other tapeworms.

Symptoms.—It is claimed that lambs and yearlings are chiefly affected; that poor growth is noted in the fall; and that by late fall and early winter the symptoms are well-marked. According to Curtice,[1] affected lambs have large heads, undersized bodies, and a stiff gait; tenesmus and diarrhea may be present, and death is due to inability of grazing lambs to withstand storms and cold.

Treatment.—In experiments with bis (5-chloro-2-hydroxyphenol) methane,* Ryff, Honess and Stoddard[4] found it to be effective in removing the fringed tapeworm, as well as *Monezias*, in dosage of 0.25 Gm. per pound of body weight for sheep on feed, and 0.05 Gm. per pound after 24 hours of starvation. Small lambs of 30 to 40 lb. required more per pound, so that a practical dosage level for lambs on feed was 0.4 Gm.

Monezia expansa.—This parasite, introduced from Europe, is common in lambs in most parts of the United States. Its habitat is the small intestine, where it may reach maturity and attain a length of 5 yards in a 2-month-old lamb. The length is from 15-30 feet, the width of the broadest segment 2 cm.; the eggs are globular or polygonal, and 50-70 microns in diameter. The end segments are filled with eggs which pass out in the feces, and their presence here serves to establish the diagnosis. Stunkard[5] has demonstrated that mites (*Galumna* spp.) are the intermediate hosts of infective larvae (cysticercoids) in the life cycle of *Monezia expansa.*

An outbreak of tape-worm disease in calves, due to *Monezia expansa*, has been described by Link, R. P., et al.[6]

Symptoms.—*Monezia* is capable of causing disease in lambs under six months old; in older sheep its presence may be ignored. Usually, where tapeworm disease is suspected, other associated parasites, such as the small intestinal worms, are the cause of symptoms. Yet most authors concede the possibility that heavy infection may cause unthriftiness, and diarrhea or constipation. As reported by the Federal

* Diphenthane-70 (20% suspension); teniatol (15% suspension); and teniathene (0.5 Gm. tablets), Pitman-Moore Co.

Bureau of Animal Industry, 1940, p. 80, experimental infection of lambs 4 to 7 weeks old persisted 40 to 81 days. The average weekly gain per animal was 1.24 pounds, as compared with 2.43 pounds in uninfected lambs. It was concluded "that tapeworms acquired by lambs early in life exert a deleterious effect on these host animals by retarding normal gain in weight."

Treatment.—The combination of nicotine sulfate and copper sulfate (1.5 per cent of each), as used for the expulsion of stomach worms (page 207), has an efficiency of approximately 100 per cent against the common tapeworm of sheep. The administration of 0.5 Gm. of lead arsenate per lamb in capsules has been reported by McCulloch and McCoy[7] as a reasonably safe and effective taeniafuge against *Monezia* spp.; a single dose was followed by the expulsion of many tapeworms, and the treated yearlings improved.

According to Brandenberg,[8] 1 pound (500 Gm.) of kamala mixed with 3 ounces (120 Gm.) of freshly powered copper sulfate is highly effective: adult sheep, 90 Gm.; 60 to 90 lb. lambs, 30 Gm.; 40 to 60 lb. lambs, 2.5 Gm.; 25 to 40 lb. lambs, 2 Gm. Prefeed on soft food, fast for 16 to 24 hours, withhold feed for a few hours after medication, then give one feeding of dry hay and turn to pasture.

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OTHER PARASITIC DISEASES OF THE SMALL INTESTINES

The following description includes a group of parasitic diseases of the small intestines frequently associated with stomach-worm disease and caused by small worms belonging to the family *Trichostrongylidae* (trichostrongyles). These diseases may occur only in certain places and only occasionally, but they appear to be increasing in frequency and importance. There may be differences of opinion concerning the patho-

genic action of the parasite or it may appear to be harmless in one section of the country and harmful in another. These parasites include various species of the following genera: *Trichostrongylus, Nematodirus, Cooperia*, and *Bonostomum*.

Trichostrongylus spp.—In the United States the most important species of this group in the small intestine is *T. colubriformis (instabilis)* (1/4-1/3 inch long). The habitat is the duodenum, occasionally the abomasum, of sheep and goats. Because of their small size, location in close contact with the mucous membrane, and covering of mucus, they are easily overlooked on autopsy. They may be recognized by placing a scraping from the mucous membrane in a shallow glass dish of water and holding it over a black background. The eggs are 75-95 x 35-40 microns and they resist drying 15 months. Freeborn and Stewart[1] state that it is not uncommon to find these parasites so plentiful as to form a hairy mat on the surface of the mucosa, that their pathogenicity is unquestioned, and that their prevalence in California is equal to that of *Ostertagia*. Experimental infestations, reported by Andrews,[2] produced a profuse diarrhea in 13 to 59 days after the first dose of larvae and caused the death of the host 3 to 53 days later. In three instances death occurred before the worm eggs appeared in the feces, thus making impossible a positive diagnosis of these cases prior to autopsy. While it is reported by Ross[3] that trichostrongyles usually do not give rise to any obvious lesions in the bowel, Andrews[2] refers to "an accumulation of serous fluid in the abdomen, diffuse inflammation of the small intestine, and a friable condition of the liver," in sheep and goats experimentally infected with known numbers of infected larvae. In animals dying of trichostrongylosis no anemia was observed. In the diagnosis of trichostrongylosis, egg counts are deceptive for they are poor layers. Emphasis is placed on the presence of black scours before or shortly after weaning, colorless or reddish fluid in the body cavities and the absence of anemia and subcutaneous edema.

Nematodirus spathiger, N. filicollis (Tread-Necked Strongyles).—This parasite is located in the small intestine, often in large numbers, from 6 to 20 feet posterior to the abomasum. The male is 1.5 cm., the female 2.3 cm. long and reddish in color. The eggs are elongated oval, 150-200 x 75-90 microns. Infective larvae may form within the egg before hatching, and infection occurs either by ingestion of the embryonated egg or the larvae. According to Hall[11] and coworkers, *N. spathiger* appears to be quite common in sheep in the United States. Welch[5] reports that in Montana "*Nematodirus* is our chief intestinal parasite. In most cases of intestinal parasitism in lambs, we find this parasite to be the principal offender." This observation is supported by a

statement in the Report of the Committee on Parasitic Diseases, United States Live Stock Sanitary Association,[6] 1937, that "observations over a period of 20 years in Montana indicate that of all the intestinal nematodes of sheep the most severe pathogenic effect is produced by *Nematodirus*, which, in the past, has been generally considered to be of minor importance. Control work on parasites should include recognition of the importance of this worm."

Cooperia curticei (sheep and goats), *C. oncophora* (sheep and cattle).—The habitat is the duodenum, occasionally in the abomasum, of ruminants. In size, location, and difficulty of finding, it corresponds with *Trichostrongylus*. The eggs are 60-80 x 30-35 microns. Fatal infections in goats from heavy infection with *C. curticei* have been reported from Australia by Edgar,[7] who observed that this parasite alone may have definite pathological significance. Severe infections in calves and sheep on wet pastures have been reported. In experiments conducted in the U. S. Bureau of Animal Industry (An. Rep. 1936, p. 54) it was observed that sheep experimentally infected with *C. curticei* required 80 pounds more feed than did noninfected sheep per 100 pounds of gain in weight.

Bunostomum trigonocephalus (Sheep Hookworm).—The habitat is in the posterior part of the small intestine; they are 1 inch long, thread-like, and reddish in color. The eggs are 80 x 40 microns. The life history is direct. This parasite is common in sheep in the Southern States and it is infrequent where there are severe frosts. Shaw[9] writes that they have been found quite prevalent in a few bands of Oregon sheep.

Nodular disease is an important parasitic disease of sheep and the lesions are occasionally found in the small intestines—see animal parasites in the large intestine for description.

Morbid Anatomy.—On postmortem examination chief interest attaches to the number and character of the worms rather than the lesions in the intestine which may be slight. In suspected parasitism the presence of fluid in the body cavities is highly suggestive. The most favorable material for examination is an animal slaughtered in an advanced stage of the disease.

Symptoms.—The symptoms are practically identical with those caused by round worms in the abomasum. In many instances both the small intestine and the abomasum are involved. As commonly described, trichostrongylosis is chiefly a disease of lambs and young sheep from the age of weaning to 18 months, but there are many exceptions. The characteristic symptoms are unthriftiness, diarrhea, anemia, loosening of the wool, pale mucous membranes and a rather high mortality

that is distributed over several weeks. In advanced cases edematous swellings in the submaxillary region are common (bottle jaw). The appetite usually remains good. The seasonal occurrence is from spring until fall.

Treatment.—For the expulsion of *Trichostrongylus* spp., the combination of copper sulfate and nicotine sulfate as administered in stomach-worm disease (page 207) is effective. *Cooperia* and *Nematodirus* may be expelled by a mixture of tetrachlorethylene and mineral oil (5-10 cc. of each) given directly after predosage with 2.5 cc. of 10 per cent copper sulfate solution. Tetrachlorethylene is also given in capsules, but the preceding method is more efficient. Phenothiazine has proved to be an efficient vermifuge against *Trichostrongylus*, but not against *Cooperia* or *Nematodirus*. Turk[8] writes that phenothiazine-salt (1:10) mixture aids in preventing a worm parasitic load from building up, but it will not remove the parasites from a heavily infected lamb, and that anthelmintics are effective only in conjunction with proper nutrition and pasture rotation.

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ANIMAL PARASITES IN THE LARGE INTESTINE

NODULAR DISEASE OF THE SHEEP AND GOAT

(Oesophagostomiasis Nodularis; Nodular Disease)

Definition.—A nodular disease of the large intestine, to some extent of the small intestine, caused by larvae and adults of *Oesophagostomum columbianum*, and characterized by diarrhea, emaciation, and anemia.

In 1910 Hall[1] reported that in the United States it was confined to the Eastern, Southern, and Middle Western States. In 1920 Ransom and

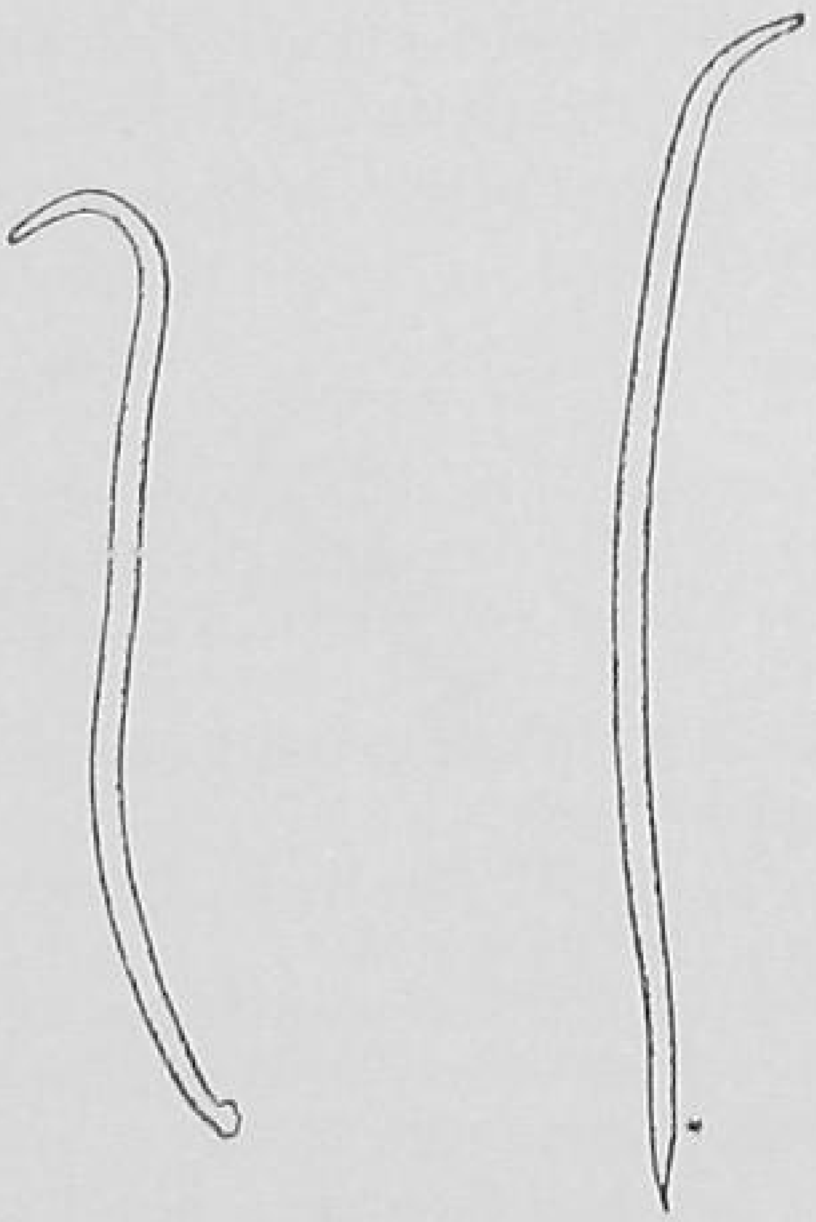

Fig. 33.—*Oesophagostomum columbianum*. Male at left, female at right. x 5. (Ransom, U. S. Dept. Agr., B.A.I., Bul. 127.)

Hall[2] wrote that it was spreading and increasing in extent and importance in the United States. The parasite seems to be native to this country. In infected areas nearly all sheep have nodules in the intestines, and this makes it difficult at times to convince the owner that the disease is serious. As a sporadic and enzootic affection it causes heavy loss by affecting the nutrition and by death. Hall has quoted Salmon as stating he believes this malady is the chief obstacle to successful sheep raising in the Southern States, and Smith and Niles as reporting instances in Virginia in which over half of large flocks have been lost

as a result of this worm. While this disease is said to be an affection of warm climates, it is widely prevalent in the cold climate of northern New England and it has been reported as common in Quebec. *Oesophagostomum columbianum* and *Haemonchus contortus* are reported by Swales[3] to be the two most important nematodes in Eastern Canada.

Life History.—The habitat of the adult is in the large intestines posterior to the cecum. The male is 10-15 mm. long, the female 14-18 mm. The eggs are 65-75 microns long by 40-50 wide. The worms have a characteristic white color which differentiates them from most of the

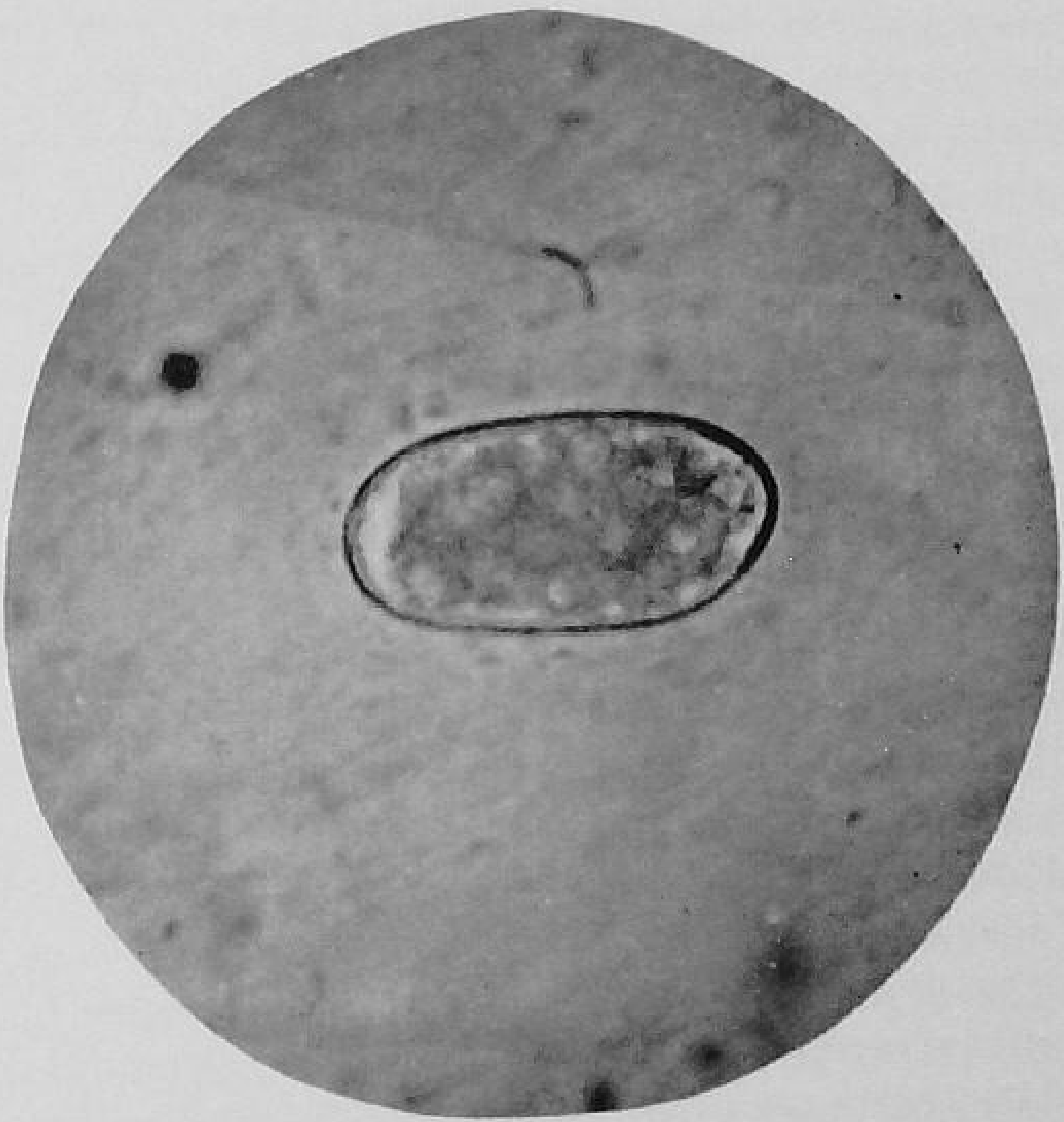

Fig. 34.—Egg of *Oesophagostomum columbianum*. (Photograph by Volgenau.)

other sheep parasites. Veglia[4] has described the life history. The development is similar to that of *Haemonchus contortus*. After the eggs pass out in the feces they become encysted and infective in about a week and are then resistant for more than a year. At night and on rainy days the larvae crawl on the grass, and on bright warm days they go down to the ground. After ingestion by lambs they immediately penetrate the intestinal wall of the large bowel, and since lambs generally have no immunity against them there is no tissue reaction and no resistance against the invasion. The larvae develop in the intestinal

wall and after a period of five days return to the lumen of the intestine as young worms; after about forty days from the date of infection eggs are passed in the feces. This development may also take place in the same manner in certain older sheep that have no resistance. Migration into the lumen of the intestine causes severe irritation of the mucosa and results in diarrhea; this is one of the chief causes of injury by the parasite. In lambs and nonresistant sheep there may be many mature worms in the cecum with no corresponding nodular changes in its wall; the nodules are rarely seen in lambs. In older sheep that possess a certain degree of immunity, penetration of the wall by larvae causes a tissue reaction and the formation of nodules which enclose the parasites. Larvae which escape from these nodules may wander about in the wall of the intestine, but usually they do not return to the lumen.

Damage is caused by injury to the muscosa during migration of larvae from the intestinal wall; by toxic secretions of adult worms which may act directly on the mucosa or by resorption; and by damage to the intestine by large numbers of nodules in its wall. In the eastern part of the United States parasitic nodules are present in the intestines of nearly all sheep, and when they are scattered so thickly as to be almost continuous over large areas of mucosa the sheep suffer from malnutrition, and perforation of the bowel may occur. Recovery is said to be rapid when the worms are removed. The adult has been found to live in the intestine of the sheep for twenty to twenty-one months in the majority of cases, and in exceptional cases even longer.

Morbid Anatomy.—The cadaver is emaciated, and the internal organs are anemic and atrophied. In both the small and large intestine are found hundreds and thousands of nodules from ⅛ to ¼ inch and even larger in diameter. Many of these are confluent, so that extensive areas of the intestine are almost wholly involved. Perforations are common. Nodules are usually most abundant on the cecum, but they may be found in the adjacent lymph glands, the omentum, and the liver. Recently formed nodules are greenish, cheesy, and may contain larvae. The older nodules are calcareous, vary in color, and are free from larvae. The nodules are rarely seen in lambs and in nonresistant sheep.

Symptoms.—Veglia[4] has described two clinical forms. The first, traumatic and septic form, develops in about a week after a severe infection, and is characterized by thirst, grinding of the teeth, persistent diarrhea, and prostration. This attack is caused by migration of the larvae into the lumen of the intestine and the mortality is high. The feces are rich in mucus and pus. Lambs often stand in a stretched out position with the hind legs and forelegs extended.

The second, toxic form, was observed in lambs one to two years old. They had resisted the initial attack, and several months later suffered from mild attacks of alternate diarrhea and constipation. They were weak and stiff. The conjunctiva and mucous membranes were not especially pale, as in haemonchosis. After a course of three or four months, when there had been loss of flesh and wool, there developed in some a pronounced muscular weakness of the hind parts.

Curtice[5] was among the first to describe the disease in the United States. He wrote that "yearlings may show considerable infection, but it is usually in the older sheep that the most abundant infection occurs." Apparently Curtice has reference to the abundance of nodules within the sheep.

While Veglia was unable to infect adult sheep as readily as lambs, it is not unusual to meet with heavily infected fatal cases in such animals; probably the infection has progressed from a younger age. An owner sometimes states that the disease did not exist in his herd until after the purchase of sheep that scoured, and from these it has gradually spread to the orginal flock. In general, it is a disease of the fall and winter. Scouring begins among the lambs in the fall and it gradually becomes worse. With a good appetite and an abundance of food 50 per cent of the weight may be lost within a month. Physical examination reveals debility, emaciation, a pale dry skin, and dry wool. Because of the disturbance of the cecum, diarrhea is severe. It has been observed in abattoirs that sheep carrying many nodules are below the others in condition. The eggs are abundant in the feces, but they are not readily differentiated from those of other intestinal parasites.

Phenothiazine has proved to be highly effective against the removal of *Oesophagostomum columbianum*. The dose for mature sheep is 25 grams. The details of administration are described under the treatment of stomach-worm disease in sheep (p. 208). Swales[3] and coworkers have reported from Canada that one early spring treatment in 1940 and a similar one in 1941 reduced the incidence of nodular disease lesions to 0.65 per lamb, a reduction from the figures of 1938 of 99.1 per cent in the lambs slaughtered in the autumn.

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NODULAR DISEASE OF CATTLE

Oesophagostomum radiatum.—The larval form of this parasite is sometimes found in cattle in the wall of the small intestine near the cecum. It is never present in sufficient numbers to cause symptoms. The nodules are $\frac{1}{8}$ to $\frac{3}{8}$ inches in diameter, and on incision present a greenish, cheesy surface. They have been mistaken for tuberculosis.

STRONGYLOSIS IN EQUINES

Definition.—Strongylosis in equines is either an inflammation or necrosis of the cecum and colon caused by adult and larval forms of

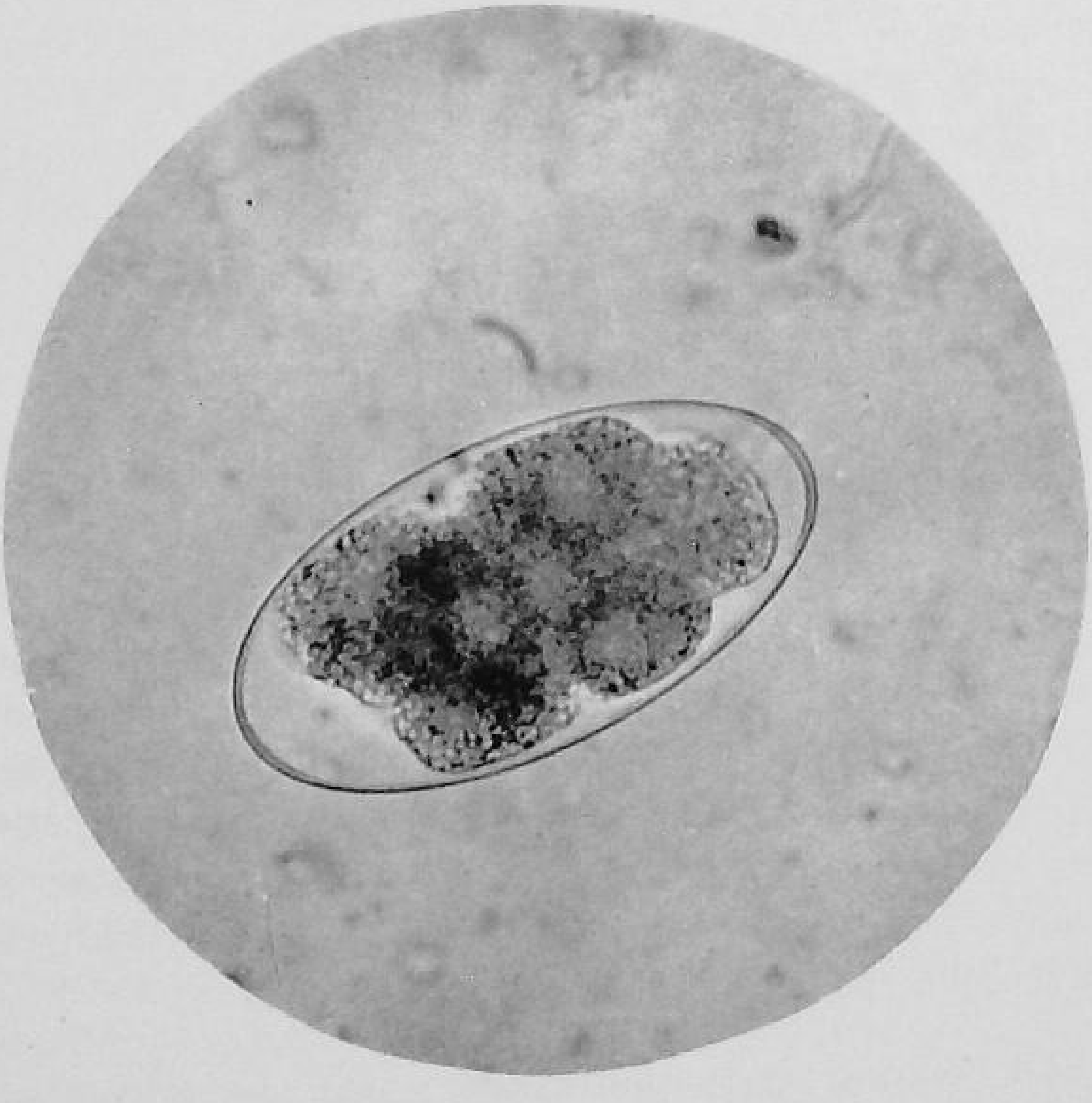

Fig. 35.—Egg of *Strongylus*, horse. (Photograph by Volgenau.)

strongyles and cylicostomes. Larval forms may also cause aneurisms, emboli, and thrombi of the great mesenteric artery and its branches, as well as inflammation of the serous and subserous tissues of the pleura and peritoneum. This is the most important intestinal parasitism of the

horse. While it affects animals of any age, it is most injurious to yearlings and 2-year-olds.

Etiology.—Parasitism in horses is especially prevalent where there is continuous grazing on heavily infected pastures. This occurs on the horse-breeding farms in Virginia and Kentucky and in the Middle West. Spreading of manure on horse pastures may also cause heavy infection of the soil. While horses of all ages harbor parasites, chief injury occurs from birth to maturity, and the amount of injury depends on the number of parasites, the age of the horse, and the state of its nutrition. Destructive infections occur when young horses drink from a heavily infected common water hole.

Description of the Parasites.—There are two chief groups: large strongyles (sclerostomes, blood strongyles) and small strongyles (cylicostomes). The large strongyles (*Strongylus*) include three species: *S. vulgaris*, *S. edentatus*, and *S. equinus*. The small strongyles include the genera *Trichonema (Cylicostomum)*, *Triodontophorus*, and several others of less importance. The adult strongyles are blood suckers, and the presence of red larvae embedded in the mucosa of the large intestine indicates that they also are blood suckers. In the colon and cecum they cause severe injury by abstraction of blood, by mechanical injury to the mucous membrane, and by the production of hemolysins.

S. vulgaris is present in nearly all equines. The habitat of the adult is the cecum and colon. The male is 15 mm. in length, the female 25 mm. The eggs are 75-80 microns long by 40-50 wide. They are deposited in the large intestine and while undergoing division pass out in the feces; they do not hatch in the intestine. Under favorable conditions of moisture and temperature hatching and further development occur outside, and in a few days the resistant ensheathed stage of the larvae is reached. They are now infectious, able to resist moisture and freezing, and in temperate climates they may survive the year round. After being swallowed in feed and water their further development is thought by most parasitologists to be as follows: From the intestine the larvae enter the circulation and pass through the liver and lungs, where some are arrested and perish. Many continue on to be deposited, chiefly in the walls of the great mesenteric artery, but to some degree in its branches. Here they cause aneurisms, thrombi, and emboli, and pass to the walls of the cecum where they form cysts or abscesses, and from which they finally emerge into the lumen as mature parasites.

S. edentatus is somewhat less common than *S. vulgaris*. Its habitat is the cecum and colon. The male is 25-33 mm. long, the female 33-36 mm. The larvae are often found under the serous membranes of the pleura or peritoneum, but they may be found in other places.

S. equinus is less common than the others. Its habitat is the cecum and colon. It is 30-40 mm. long.

Trichonema (Cylicostomum) includes a number of species, and no useful object is served in attempting to diagnose all the species present as they are never found in pure culture. The size varies from 5 to 15 mm. in length, according to the species. The habitat of the mature parasite is in the cecum and colon, and a part of its life cycle is passed in the walls of the intestine where the larval form is a frequent cause of serious injury.

Morbid Anatomy.—The cadaver may be normal or greatly emaciated. The body cavities contain clear or reddish serum whenever many larvae are present in the serosa and subserosa. The peritoneum presents areas of hyperemia and necrosis, and in these areas larvae may be found. Larvae may also be abundant in the pleura and diaphragm. Verminous thrombi are invariably found in the trunk of the anterior mesenteric artery, and these may contain many strongyles. Occlusion of mesenteric blood vessels results in necrosis of the corresponding intestine. Usually this is the cecum, but it may be the small intestine. The necrosis may be in the form of circumscribed areas, as seen in the cecum, or affect a segment of 10 to 20 feet of the small intestine. The wall is thickened, yellowish or black in color, and friable. Occasionally the wall of the colic artery is thickened and filled with abscesses through the greater part of its length, and the lumen may be very small *(arteritis hyperplastica, a. obliterans)*. In the cecum and colon, both free and in the walls, one may find hundreds and thousands of red worms in a horse dead of verminous enteritis. Abscesses containing larvae of strongyles are also found in the walls of the large intestine. There may be congestion and swelling of the mesenteric lymph glands.

Symptoms.—Infection is most abundant when colts drink from stagnant pools. Destructive enzootics may develop in six to twelve months after such exposure. The presence of many larvae in the mucosa gives rise to severe irritation, enteritis, and general disturbance.

FORMS OF THE DISEASE.—These vary widely according to the location of the injury and the degree of infection. In all, there is a history of unthriftiness, poor appetite, and weakness.

(a) Intestinal Form with Diarrhea and Marked Debility.—This type occurs as an enzootic in colts and young horses that have received a heavy infection. Emaciation, anemia, and weakness are marked. Colts become so weak that they cannot rise without aid. The hair is long and the conjunctival mucosae are pale. Febrile symptoms are usually absent, yet when the serous membranes are affected (pleuritis, peritonitis) there may be a rise in the pulse, respiration, and tempera-

ture. The abdomen is small or pendulous—"pot bellied"—and the peristalsis is usually suppressed. Evacuations may be scanty, but more often there is an uncontrollable diarrhea; the latter condition results from the presence of many larvae, especially Cylicostomum, in the mucosa of the large intestine. On rectal examination it may be possible to feel an aneurism of the trunk of the anterior mesenteric artery in the form of a firm pulsating swelling. The intestinal symptoms vary according to the location of the chief lesions. When the colic artery is extensively thickened (arteritis), or the larvae are chiefly in the subserosa, diarrhea may be absent. When an embolus causes necrosis of the cecum, or when larvae are numerous in the walls of the colon and cecum, diarrhea is persistent. A few suffer from attacks of colic. The course is over a period of a few weeks and the mortality is high.

(b) Verminous Colic (Thrombo-Embolic Colic).—This occurs in adults and colts. Affected animals are subject to repeated colic that comes on during exercise, and has no dietetic origin. Usually it is caused by the presence of emboli or thrombi in the terminal branches of the mesenteric artery (thrombo-embolic colic). The degree of passive congestion or necrosis of the bowel depends on the extent to which the circulation is cut by the plugging of the vessels. This is seldom so complete as to cause death, but the attack is severe while it lasts. The course is from one to twenty-four hours. Verminous colic does not appear to be frequent in horses in the northeastern part of the United States. Two fatal cases reported in our ambulatory clinic revealed necrosis of the ileum in well-nourished adult horses. Other suspected subjects were poor and tired easily. A report from Morris[1] of the Louisiana Experiment Station indicates that verminous colic is common among horses and mules in that State, and that apparently it is caused by parasitic invasion of the mucosa as well as the blood vessels. In a series described by Williams[2] the most frequent form was a sudden attack of severe and continuous colic with death within twenty-four hours, then followed recurrent or prolonged colic, while a third group became unthrifty and finally died of colic.

(c) Paralysis of the Hind Limbs is caused by a thrombus of the iliac arteries. A horse affected in this manner has a history of becoming lame or falling down when driven. Under exercise, a lameness develops in one or both hind legs, and loss of control soon follows. On standing, the affected leg may be held in an abducted position; the muscles tremble, and sweating spreads from the limbs to the body. After a rest of a few minutes to a few hours the symptoms disappear.

(d) Unthriftiness.—The presence of many adult parasites, especially cylicostomes, in the large intestine is a frequent cause of unthriftiness

in horses and colts. In spite of a good appetite and sufficient food, they become poor and sweat and tire easily when worked. The hair is long and dry and the mucous membranes are pale. Depraved appetite and rubbing the tail are often observed. These symptoms are most prominent in the spring when the horse is worked hard while green; such animals may show muscular twitchings and even convulsions after a few hours work. Diagnosis is confirmed by the presence of many eggs in the feces.

Treatment.—Phenothiazine is perhaps the best anthelmintic yet discovered against strongyles in the large intestines, but there have been occasional reports of fatal toxic action. Apparently these are the result of medicating debilitated horses, or horses on a low protein diet. An example of undesirable reaction from medication of debilitated and underfed horses has been reported by Fincher and Gibbons.[3] Folse[4] has reported excellent results from the use of phenothiazine, even in large doses, to horses on alfalfa hay, and fatal complications in another group fed upon sorghum hay; but the sick animals recovered rapidly following blood transfusion. Errington[5] observed reactions in two of four thoroughbred stallions in excellent condition that were fasted approximately 24 hours and then given 90 Gm. of phenothiazine. On the following day there were inappetence, almost black urine, jaundice and decreased peristalsis; on the third day both horses had mild colic and on the sixth day both were normal. He advises a dosage of 30 Gm., which seems to be near the minimum efficient dose for animals the size of mature thoroughbreds. Knowles and Blount[6] found it to be nontoxic in therapeutic doses of 30 Gm. (0.066 Gm. per kilogram), and 100 per cent efficient in the treatment of equine strongylosis; they report that it supersedes all other forms of treatment for strongylosis in equines. It may be administered in suspension, in a capsule, or in the feed. It should be given without a preliminary fast; it is not a purgative and need not be followed by a purgative. To avoid toxic action in debilitated animals administer small doses of about 3 Gm. daily.

In 1949 Dimock[7] reported that a daily dose of 2 Gm. phenothiazine stopped strongyle egg production and eliminated the small nematode parasites of the large intestine, but had little or no effect on the ascarids. And in 1950 Todd et al.[8] wrote that strongyle infections have been reduced in four groups of horses fed 0.5, 1, 2, or 4 Gm. daily for two years in proportion to the amounts fed, and without harm. Sixteen yearlings carrying extremely heavy counts of strongyle eggs in the feces received 30 grains of phenothiazine in the feed once daily for twelve weeks, when the control of strongyles was complete.[9]

The use of noninfected pastures, rotation of pastures, and weekly

removal of feces from the pastures of breeding studs reduces the infection.

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OTHER PARASITIC DISEASES OF THE LARGE INTESTINE

Oxyuriasis

Oxyuris curvula (*equi*) (Pin-worm, Thread-worm) is common in the rectum and colon of equines. The female is most abundant and measures 4-15 cm. in length. The anterior part of the body is thickened and curved; the posterior part is long and pointed, somewhat thread-like. The ova are elongated, about 90 microns long by 40 microns wide, with a lid-like formation at one end. The eggs are deposited around the anus where the females are attached by their thread-like tails. Their presence here may cause itching, and rubbing of the tail with loss of hair. Yellowish masses formed of eggs and mucus may be deposited on the skin of the anus. The adults are harmless inhabitants of the cecum and large colon.

Treatment.—The worms may be easily removed from both the rectum and colon. Hall[1] has reported complete success with each of the following methods: Oil of chenopodium (16 cc.) followed immediately with a quart of linseed oil, the horse being fasted 36 hours previous to medication; or turpentine (60 cc.) followed immediately with a quart of linseed oil, the horse being fasted less than 24 hours previous to medication; or tartar emetic (8 Gm.) daily in the feed for five days. The writer has seen hundreds of these worms expelled from a horse by

a single dose of aloes 1 ounce (30 Gm.). Pin-worms may be easily removed from the rectum by an enema of soap and water, or 1 per cent solution of creolin, or an infusion of quassia chips, or a 1 per cent solution of acetic acid, or a mixture of vinegar and water. Habermann et al.[2] found that phenothiazine was ineffective for the removal of *Oxyuris* sp.

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Whipworm

Trichuris ovis (Whipworm) is a common parasite in the cecum in sheep and goats. It may cause trouble when present in large numbers, but this is infrequent. The female is 5 to 7 cm. long, the male 5 to 6. The anterior portion of the body of this worm is very slender and two to three times the length of the thick posterior portion. The common name, whipworm, is derived from its resemblance to a whipstock and lash. The life history is direct, without intermediate host. No definite symptoms have been ascribed to the effect of this parasite, but it is assumed that it may affect the health of the sheep when present in sufficient numbers. No effective vermifuge has been found.

DISEASES OF THE PERITONEUM

PERITONITIS

Etiology.—Inflammation of the peritoneum is the result of various causes: (*1*) Traumatic gastritis is by far the most frequent form in animals. (*2*) Puerperal peritonitis in all species is next in importance. (*3*) Traumatism, from a blow on the abdominal wall, or a perforative wound, may cause peritonitis. In this group are those cases that follow castration, hernia operation, and manipulations in the rectum. (*4*) In colts heavy infection of larvae of *Strongylus edentatus* is an occasional cause. (*5*) Chronic specific infection, as tuberculosis, necrobacillosis, and actinobacillosis has been observed. (*6*) Acute general infections, as anthrax, hog cholera, diseases of the newborn, septicemia, and acute enteritis often include the peritoneum. (*7*) Intestinal obstruction in the form of displacements, parasites, food impactions, foreign bodies, strangulated umbilical hernia, and enteroliths occasionally result in necrosis with rupture of the intestine and peritonitis. (*8*) In two cows observed in the ambulatory clinic, an acute diffuse peritonitis followed perforation of the abomasum by a caustic poison, said by the chemist to be calcium cyanide. The gastroenteritis and peritonitis were intense. In another, what appeared to be infection of the mucosa of the abomasum with *Actinomyces necrophorus* led to involvement of the adjacent rumen with a 3-inch rupture of its wall and peritonitis; this was diagnosed as traumatic gastritis. Chronic adhesive peritonitis resulting from perforation of an ulcer of the abomasum, and from an abscess directly over the umbilicus, has been observed in cows. Eveleth and Hilston[1] have described peritonitis in sheep due to rupture of the intestine in nodular disease. In a horse a chronic abscess on the abdominal floor led to rupture of the sternal flexure of the colon; the temperature was 106° F. Rupture of the bladder also results in peritonitis.

Morbid Anatomy.—*Chronic circumscribed* peritonitis, the most frequent form, is found in chronic traumatic gastritis, in recovered perimetritis, and after operative and trocar wounds that enter the peritoneum. *Acute circumscribed* peritonitis is found after intestinal torsion and similar displacements. *Chronic diffuse* peritonitis results from the specific chronic infections, especially tuberculosis, and occasionally from traumatic gastritis. *Acute diffuse* types are associated with traumatic gastritis, perforative gastritis from caustic poisons, metritis, and acute general infections.

In Beaver's[2] abattoir case of *actinomycosis* in a well-nourished carcass, the omentum was adherent to the rumen and contained many nodules 1-3 mm. in diameter. These were covered with thick hard

capsules over caseous material without calcification. In *tuberculous* peritonitis in cows the omentum may be uniformly thickened by many small, calcified nodules; there may be extensive adhesions and nodular growths over the parietal peritoneum and the rumen; salpingitis is a frequent associated lesion, and generalized tuberculosis is the rule.

In necrobacillosis the peritoneal cavity contains yellow serum, adhesions are extensive, and in one case the intestines were embedded in a yellow gelatinous mass. It is commonly associated with pleuritic adhesions. The omentum presents a peculiar brownish yellow color.

Symptoms.—Acute circumscribed peritonitis, as it occurs in traumatic gastritis, is described under that title. In cows there are two acute diffuse forms that may closely resemble each other—puerperal and traumatic. The former is associated with advanced pregnancy or recent delivery, which implies regard for the general rule that an acute attack of any kind at this time is parturient in origin. The parturient type is somewhat more sudden in onset. There is a degree of stiffness and reluctance to move not commonly approached in traumatic gastritis. Perforation of an infected uterus often terminates in death within three days, while perforation of the reticulum may not prove fatal until ten to fourteen days. In both, one finds dullness and rapid emaciation with a tendency to remain down. The pulse is high, the breathing is shallow and irregular, and with few exceptions the temperature is normal or only slightly raised. Painful expression and attitude, as well as pain on percussion over the abdomen, are always present. In peritonitis in the horse the pulse and temperature gradually rise.

In subacute and chronic forms (necrobacillosis, tuberculosis, perforation of the reticulum), a high fever and pulse in a young bovine are suggestive of necrobacillosis. Intussusception in cows may be diagnosed as peritonitis. Perforation of the abdomen of a horse is followed by dullness, sweating, weakness, suppressed peristalsis, a high pulse, shallow breathing, and medium fever. There is a more or less prevalent opinion that in indigestion in horses and cattle, peritonitis is apt to develop, but a review of autopsies shows that deaths in horses are mainly from gastric dilatation or impaction of the colon, often with rupture, and that deaths in cattle are mainly due to gastroenteritis. With the exception of certain forms of metritis, and traumatic peritonitis, the course is not affected by treatment.

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DISEASES OF THE LIVER

JAUNDICE

Definition.—Jaundice or icterus is a staining of the tissues and fluids of the body with bile-pigment. It is a symptom of various abnormal conditions. Two chief forms are recognized in domestic animals: (1) Obstructive hepatic jaundice in which the bilirubin has passed through the liver cells and then been absorbed; (2) Toxic and infective hepatic jaundice.

Etiology.—Among the causes of *obstructive* hepatic jaundice are: (*a*) Obstruction by foreign bodies within the ducts, as parasites (flukes, ascarids); (*b*) by duodenal catarrh, either primary, or secondary to impaction of the colon in the horse; (*c*) by pressure upon the duct from without, as by tumors, tubercles, necrotic nodules of the liver in cows, and other rare causes. The main causes of *toxic and infective* hepatic jaundice are: (*a*) chemical poisoning, as from arsenic, lead, copper or phosphorus; (*b*) toxic conditions as fatty degeneration of the liver, azoturia and hepatic cirrhosis; (*c*) infectious diseases, as severe equine influenza, equine pneumonia, Texas fever, anaplasmosis; and (*d*) post-parturient hemoglobinuria, bacillary hemoglobinuria, and in most affections in which there is rapid hemolysis.

Symptoms.—The distinctive symptom is a yellowish discoloration of the mucous membrane and the white parts of the skin. In most animals this is best seen in the conjunctival sclera. In well-marked obstructive jaundice, as in impaction of the colon in the horse, the urine may be dark or black, as in azoturia. The other symptoms and the prognosis depend on the nature of the primary disease. In catarrh of the bowel, recovery is the rule; in infectious diseases the degree of jaundice indicates the degree of hemolysis. In swine a yellowish discoloration of the skin indicates the presence of ascarids in the bile ducts. In Bonnikson's[1] case of a mare with larval obstruction of the bile duct, the mucosa was green; in human medicine this is termed "black jaundice." In general the degree of icterus bears a direct relation to the severity of the disease, whether it be impaction of the colon, parasitic invasion, or an infection. In disease of the liver, changes that are diffuse and taking place rapidly tend to cause jaundice, while a more circumscribed and less active process of the same general character may cause no discoloration. The color varies from a tinge of lemon-yellow to a deep orange. The condition is most intense in severe affections of the liver, as fatty degeneration in cows, liver fluke, and cirrho-

sis. A combination of jaundice and brain symptoms in domestic animals indicates primary disease of the liver.

In chronic copper poisoning in sheep, symptoms described by Boughton and Hardy[2] were depression, trembling, jaundice, pulse from 120 to 160, respirations 40 to 60 and shallow, and temperature normal (102 to 104). There was a profuse nasal discharge of blood and mucus, and the urine was port wine color. Death occurred in from twenty-four to forty-eight hours. This condition resulted from the long-continued ingestion of commercial salt mixtures which contained relatively small percentages of copper sulfate in addition to sodium choride and tobacco dust.

A peculiar form of enzootic toxemic jaundice in sheep and cattle in Australia was described by Chamberlin[3] in 1933, and by Rose and Edgar[4] in 1936. The outstanding characteristics were an intense icterus associated with hemoglobinemia and hemoglobinuria. A hemolytic toxic bowel filtrate was recovered from cases in both sheep and cattle. It is considered to be an enterotoxemia caused by infection with *Cl. welchii*, type A.

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FATTY DEGENERATION OF THE LIVER

Primary fatty degeneration of the liver is met with occasionally in pure bred cows under heavy feeding and production. The *symptoms* develop gradually. There is a history of recurrent dullness. At pasture a dummy-like sleepy attitude may be observed, especially if the weather is hot. Finally, deranged consciousness is more marked and continuous. In the stanchion the animal may suddenly jerk back in an involuntary manner or show some other unusual sign of motor irritation. In a hot stable there may be marked loss of consciousness, with various degrees of motor irritation, such as tremors, throwing the head, violent involuntary kicking, and inability to rise.[1] In a case described by Townsend[2] the onset was much like that of milk fever; this was followed by anorexia, salivation, and pain on percussion over the liver. The general symptoms are a rapid loss in condition and marked icterus

of the mucous membranes. The pulse and temperature are somewhat irregular, but there is no well-marked fever. The course is several weeks, during which time the patient is alternately better and worse. It is invariably fatal and treatment is without effect. Clinical diagnosis is based on the combined *nervous symptoms and icterus.* When the liver is greatly enlarged one may recognize an increased area of dullness, or an increase in size on rectal examination.

Fig. 36.—A case of fatty degeneration of the liver showing drowsiness and unnatural position of the front limbs. A few minutes before this photograph was taken the cow was down in a hot shed, semiconscious and suffering from clonic spasms bordering on convulsions. On being moved to a cooler outdoor place she was able to rise. Death occurred several weeks later. A few years later a second case developed on the same farm.

Morbid Anatomy.—The liver is greatly enlarged; in Townsend's case it weighed 45 pounds, was degenerated, and of an intense yellow color. The mucous membranes and the internal fat are yellow. Histological examination reveals intense fatty degeneration of the liver cells.

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INFECTIOUS HEPATITIS

Definition.—Acute infectious hepatitis in the horse is usually a postvaccinal, sometimes spontaneous, disease characterized by jaundice,

mania, forced movements such as circling or constantly pressing forward, and coma. Death usually occurs within 24 to 48 hours.

Etiology.—A fatal type of parenchymatous hepatitis in the horse was first described in 1918 by Theiler[1] in South Africa where it was termed "staggers," and where it followed the injection of "horse sickness" virus or serum, or liver emulsion of a "staggers" horse, or infusion of blood from a horse immune to "staggers" virus. In the United States this disease was first reported in 1933 in Utah by Madsen[2] following the prophylactic use of anti-encephalomyelitis serum. This was followed in 1936 by a similar report from Montana by Marsh[3] where over 5000 horses had been immunized, using 2 cc. of guinea-pig brain tissue vaccine and 50 cc. of anti-encephalomyelitis serum. Within 40 to 70 days after immunization there were 89 cases and 79 fatalities. It has also been reported from several states, always subsequent to an epizootic of infectious equine encephalomyelitis and always confined to areas previously invaded by the latter; some cases occurred in animals with no history of having received any biological product, and some few in animals recovered from frank encephalomyelitis—Shahan et al.[4]

Again in the 1940's the disease was reported from various localities in New York State following the use of tetanus antitoxin, and one spontaneous case was seen by the writer in an unused horse at pasture, where it was diagnosed as rabies. These attacks were identified through their resemblance to rabies and the shipment of material to the diagnostic laboratory; they have been attributed to foreign heated protein given as tetanus antitoxin. And more recent reports have indicated that the disease occurs in race horses that have received various immunizing products. According to an unpublished report, pregnant mare serum, Gonaden, administered to sterile mares causes infectious hepatitis.

Symptoms.—Precursory symptoms reported by Theiler[1] were dullness, stumbling, unsteady gait, anorexia, resting the head on the manger, and a peculiar vacant stare. Usually the conjunctival mucosa is yellow, and the temperature is normal with a tendency to subnormal. The violent symptoms may be preceded by colic, repeated micturition and profuse sweating, but usually they are present from the very beginning, and increase rapidly in intensity. One of the most distinguishing symptoms is the persistent tendency to forced forward movements. With the head against the wall or in the corner; this pushing forward is so violent that in a box stall the support with the head is lost and the horse shoots along the side of the wall into the opposite corner then repeats the manoeuvre, and sometimes falls in unnatural positions. After falling there are constant and violent attempts to rise. In this

manner the head becomes bruised and cut. In an open field or pasture the horse moves blindly forward or in circles through fences or other obstacles among which it may become entangled and cast; after which it makes violent efforts to rise and frequently bangs its head against the floor or ground. The mouth hangs open and there may be prolapse of the tongue. Towards the end dyspnea is pronounced and extreme sweating may be present throughout the entire course. In some cases the nervous symptoms are in the form of stupor, and an interval of somnolence may be preceded and followed by violence. With few exceptions food and water are refused from the onset. In one case observed by the author, the animal became quiet for a time, ate grain and drank water, and then resumed uninterrupted movements which continued until a few minutes before death. Theiler[1] mentions a less violent semi-conscious form in which the animal may stand with the head hanging for a long time, the legs crossed or spread apart, the gait swaying or staggering and the eyes half-closed. The pulse is normal until near the end. The most distinguishing features are mania with uncontrolled and violent movements combined with jaundice. In cases of recovery the symptoms are less intense. As observed in Montana, the predominant symptom was excitement in contrast to the sleepy symptoms of encephalomyelitis, and the mortality was 90 per cent. The affected animals walked continuously and pressed against objects such as fences. There were trembling, blindness, profuse sweating and absence of fever. The mucous membranes were yellow, and at the onset hematuria was sometimes present.

Pathology.—Except in unusual cases of sudden death, or where the comatose stage was dominant, abrasions and wounds are present on the head, while general lacerations from wire are common in horses that die in the field. The mucous membranes of the mouth and eyes are yellow, and on section the tongue may be unusually yellow. The serosa of the large body cavities may be yellow but they contain no exudate. The lymph glands may be edematous, hyperemic or hemorrhagic. The endocardial lining and valves may show a yellow tinge. The main changes are found in the liver which may or may not be reduced in size. The consistency is usually firm, more rarely it is softened or brittle. The color is light brown or light yellow, rarely dark. The capsule frequently appears pitted. Under the microscope in the central portion of the lobule around the central vein, there are as a rule no more intact liver cells to be seen. Lobules in which no liver cells can be recognized are frequent. There seem to be transition forms, from lobules in which practically all cells show fatty degeneration, to lobules in which there is little or no fat

left. To summarize, there is general icterus; pigmentation, fatty degeneration, and necrosis of the liver cells and fatty degeneration of the myocardium, and muscle.

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CIRRHOSIS OF THE LIVER

(Chronic Productive Interstitial Hepatitis; Necrobiosis et cirrhosis hepatis enzootica—Van Es)

Definition.—Cirrhosis of the liver is an increase in the connective tissue combined with a degeneration of the liver cells in which the organ may be increased in size (hypertrophic) or diminished (atrophic). Chief attention has been given to various enzootic types that are endemic in various parts of the world, but it is not rare to meet with sporadic forms of unknown etiology. It is characterized by jaundice, deranged consciousness, and motor irritation.

Etiology.—In 1892 Schroeder,[1] and Smith[2] described cirrhosis of the liver, "bottom disease," among horses in the lower Missouri Valley. It had been endemic there for many years in the late summer and fall in pastured horses. The cause was undetermined. In 1925 Kalkus[3] and others described hepatic cirrhosis, "walking disease," among horses in the Pacific Northwest. Affected areas were in high dry altitudes, and the disease followed dry seasons after which the horses were obliged to exist on dry stubble and straw stacks: the following spring many cases developed, finally disappearing in the fall. In 1893 Johnson[4] described an enzootic of hepatic cirrhosis, "Pictou cattle disease," in pastured cattle in Nova Scotia, where farmers attributed it to eating ragwort (*Senechio jacobeus*). In 1900 Gilruth[5] described hepatic cirrhosis, "Winton disease," in horses and cattle in New Zealand. Here, also it was attributed to ragwort, and Gilruth caused cirrhosis in two 6 months old calves by feeding *S. jacobeus*. In 1906 Robertson[6] reported the presence of a common chronic atrophic hepatic cirrhosis, "stomach staggers," in pastured horses and cattle in Cape Colony, and he produced the disease by feeding Senechio. In Erie County, New York, in

the Tonawanda Creek Valley, horses have suffered each winter from a hypertrophic cirrhosis that is said to affect only those fed heavily on alsike clover, and a few sporadic cases have been encountered in the vicinity of Ithaca, N.Y. In 1929 Van Es[7] and others gave a complete report on "walking disease" *(Necrosis et cirrhosis hepatis enzootica)* in Northwestern Nebraska, where it caused heavy losses among horses and to some extent among cattle, in the months of June and July. The cause proved to be *Senechio Riddelli.* In Ontario, Canada, cirrhosis of the liver caused by feeding alsike clover raised on clay soils has been described by Schofield.[8] Murnane and Ewart[9] have reported hepatic cirrhosis caused by whitewood (*Atalaya hemiglauca*), in which saponin proved to be the toxic substance. Cirrhosis of the liver in steers in Florida caused by eating *Crotalaria spectabilis* has been reported by Sanders[10] and associates. The seeds of yellow tarweed (*Amsinckia intermedia*) have been reported by McCulloch[11] as a cause of enzootic hepatic cirrhosis, known as walking disease of horses and hard liver disease of swine and cattle, as it occurs in certain regions of the Pacific Northwest.

Morbid Anatomy.—In the hypertrophic form the liver is reddish yellow or bronze and the consistency is brittle. The enlarged and brittle livers from affected horses in the Tonawanda Creek Valley, New York, have weighed as high as 50 pounds. In the atrophic form the color may be grayish or blue and the consistency hard and leathery. Van Es states, "The dominant characteristics of the morbid process are the necrobiosis of the parenchyma and the proliferation of the connective tissue elements. One or the other may predominate, but our observations tend to indicate that they develop simultaneously with parenchymatous intoxication as the initial factor. In the cases which occur late in the season, or which have survived for several months, the interstitial lesions are the most prominent."

Symptoms.—Because of the variety of causes that may operate to produce the sporadic form, the symptoms vary widely. If the case is observed carefully, however, the combination of jaundice and nervous symptoms nearly always appears. There is a history of gradual loss in condition, anorexia, debility, alternate diarrhea and constipation without apparant cause, and a possible history of attacks of colic; often the subject is young. Jaundice is present from the beginning but later it may disappear. Usually there is no fever, yet a temperature of 106° F. has been observed. Among the brain symptoms are *deranged consciousness,* such as marked dullness and somnolence, or delirium with violent destruction of the stall. Or *motor irritation* may develop in the form of muscular twitching, walking aimlessly and in circles, press-

ing the head against objects, etc. In advanced cases there may be paralysis, the horse knuckles at the fetlock or is weak behind, or is unable to swallow, or goes down and rises with difficulty. Kalkus[3] mentions a perverted appetite, and Van Es[7] the chewing of mangers and fences. In marked enlargement of the liver, one may recognize an increased area of dullness on percussion, and on rectal examination it may be possible to palpate the thickened posterior border of the enlarged and hardened organ in the right sublumbar region. Acute Senechio poisoning in horses in Nebraska, presenting symptoms similar to those of encephalomyelitis, has been described by Carpenter.[12] The symptoms were jaundice, excitation, paresis, pendulous lips, and in some instances a fever.

In one instance a 2-year-old colt treated in the ambulatory clinic developed a paralysis that was thought to result from an affection of the brain; disease of the liver was not even suspected during the animal's life. Another was treated repeatedly for impaction of the colon. An alternate diarrhea showed clay-like feces; the evacuations resembled those of a calf affected with white scours. A period of marked dullness and jaundice at the onset led to a diagnosis of disease of the liver; after about eight months, an autopsy following impaction and rupture of the colon revealed cirrhosis of the liver to be the primary disease. The course of the disease is over weeks and months and often it is remittant.

Treatment.—No effective remedy has been found. Robertson[6] states that in the enzootic form in Cape Colony recovery may occur if the animal is stabled early and fed abundantly. In the less intense forms, it is probable that the condition will be benefited by an easily digested laxative diet composed in part of molasses, and supplemented by occasional courses of bitters and salts.

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NECROBACILLOSIS OF THE LIVER

(Necrotic Hepatitis)

In necrobacillosis of the liver, circumscribed, dry, brownish-yellow nodules from ½ to 2 inches in diameter are scattered through the organ. When the disease has advanced far enough to give rise to symptoms, there are also peritonitis and nodular disease of the lungs, and in some instances the spleen, kidneys, heart and other tissues are involved.

Etiology.—*Actinomyces necrophorus (B. necrophorus)* is the specific cause. Infection enters through the circulation, and a study of individual cases reveals in some probability that it is pyemic from metritis or foot-rot or umbilical infection.

Morbid Anatomy.—A few necrotic foci in the liver do no apparent harm; they are often found in animals that have died of another disease. In those that have died from the infection, peritoneal adhesions are commonly present and one may find acute peritonitis. Occasionally the liver is enlarged. On its surface are many round, raised, dark-yellow areas an inch or more in diameter. These nodules are scattered throughout the organ. On section, they have a thin capsule and a uniform, clay-like dry consistency. Necrosis proceeds at the periphery of the nodule, where the organism is abundant and active. Other observed associated lesions are subperitoneal emphysema, distension of the gall bladder with bile from pressure by the nodules on the bile duct, necrotic foci in the pleura, lungs, diaphragm, heart muscle, spleen and kidneys, and ulcers on the prepuce.

Symptoms.—The disease is somewhat common in cattle, where it causes considerable loss; it is less frequent in horses and sheep. It is usually sporadic, but may be enzootic. Young cattle, from 2 to 3 years of age seem to be most commonly affected. In cows there is a history of

lessened milk flow, depression, and anorexia over a period of from two to ten days. In well-marked types, one often finds grunting, arched back, stiffness, and a tendency to remain down. Others show merely a failing appetite and condition. The mucosae are usually unchanged, but when the liver lesions extend rapidly there may be jaundice. The pulse is 80-100, the respirations 30-40, and the temperature 104°-106°F. High temperature and pulse are regularly present and are probably due to toxins formed by the necrosis bacillus. Towards the end of the course the temperature may fall, while the pulse and respirations rise. The abdomen is gaunt, the peristalsis suppressed, the feces slight and covered with mucus. Percussion over the liver may be painful, and in involvement of the peritoneum or pleura pain may be extensive on both sides. Because of the frequency of associated lung lesions, fast breathing, slight nasal discharge, cough, and pain on percussion over the chest are sometimes present. The course is usually two to three weeks after the first symptoms appear. The most distinctive signs are a high temperature and pulse combined with the general symptoms of traumatic gastritis. Brain symptoms have been observed in one cow that died after a sickness of four days from apparent encephalitis; the hepatic necrosis was extensive, and jaundice was either absent or overlooked. *Treatment* is without effect. After the symptoms are distinct, death occurs within a period of approximately two weeks.

ABSCESS OF THE LIVER

Etiology.—Abscess of the liver occurs under the following conditions: (*a*) *Traumatic gastritis* causes infection of the liver, either by extension from the peritoneum, or from direct injury by the foreign body. Involvement may be extensive in the form of a single large abscess, or there may be multiple abscess formation with communicating fistulae.

(*b*) *Metastatic* embolic or pyemic abscesses are frequent in severe metritis and mastitis, and in navel-ill; they are occasional in tuberculosis and actinomycosis, and are not rare in strangles. While infection from the intestine through the portal vein may cause occasional hepatic abscess, it does not give rise to symptoms.

Morbid Anatomy.—Single or multiple small abscesses are often found in fat cattle slaughtered for beef; they contain yellow creamy pus, are sharply circumscribed by the abscess wall, and injure only the affected parts. The nature of other abscesses, as in tuberculosis, is usually revealed by the associated changes in other organs. Bacteriological examination of hepatic abscesses from cows autopsied in the ambulatory clinic have yielded *Ps. pyocyaneus*, *Corynebacterium pyo-*

genes, and *Actinomyces necrophorus*. *A. necrophorus* was observed by Newsom[1] to be the cause of most liver abscesses observed in slaughtered cattle in Denver.

Symptoms.—Extensive involvement of the liver may cause the general symptoms of a chronic debilitating disease with a gradual loss of condition. In a case of tuberculous abscess of the liver examined by the author, pain on percussion was distinct, and this affords the only direct evidence of the disease. There is no increased area of dullness, and the organ cannot be reached per rectum.

Multiple abscess of the liver in cows has been diagnosed and operated upon for traumatic gastritis, and in one animal with multiple hepatic abscess impaction of the abomasum was suspected.

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LUPINOSIS

This is an intoxication, usually acute, caused chiefly by eating seed-containing pods of the chick-pea (*Lupinus*). It is characterized by nervous symptoms, jaundice, and acute yellow atrophy of the liver. In the United States it has been reported from Montana and in Europe from Northern Germany. Sheep and horses are most susceptible, though it may affect cattle and other animals.

Etiology.—Lupines grow abundantly in the foot hills and mountain ranges of Montana, where they are used for both grazing and hay in the absence of other roughage. The seed-containing pods are especially toxic. Sickness is usually traced to hay harvested before the seed-pods ripen and split, or to foraging when very hungry upon lupines that still carry unbroken pods. The nature of the toxic substance is unknown.

Morbid Anatomy.—In acute poisoning the stomach of a sheep contains pods and seeds of lupines. The small intestines are hemorrhagic and the mucosa of the digestive tract is yellow. The liver is degenerated, the gall bladder distended and its mucosa is swollen and congested. The kidneys and bladder are congested. General icterus is often present. In the chronic form in the horse, the cadaver is emaciated, and the subcutis is yellow. The disease produces a chronic progressive fatty degeneration of the liver, which is described as brown or yellow, leathery, irregularly thickened, and shrunken. The changes vary somewhat according to the duration of the disease.

Symptoms.—The *acute form in sheep* has a sudden and violent onset within 2 to 4 hours after eating the poison seeds and pods. The dominant

symptoms are deranged consciousness, motor irritation, and jaundice. The sheep run blindly into various objects, develop spasms and convulsions, and die in from one to two hours. A few live from two to four days. Wilcox[1] reports 100 deaths in 200 sheep, which were driven in a hungry condition and ate nearly ripe lupines. On one ranch 2 tons of lupine hay fed to 2000 sheep caused 700 deaths within 48 hours.

The *chronic form in horses*, described by Knowles,[2] begins with attacks of vertigo, loss in condition, and jaundice. The course is from one to twelve months, usually terminating in death.

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HEPATIC DISTOMIASIS

(Fascioliasis; Liver Fluke Disease; Liver Rot)

Definition.—An acute or chronic disease of the liver and bile ducts, caused chiefly by *Fasciola hepatica,* and often marked by destruction of the liver—liver rot. In 1910 Hall[1] reported this disease of importance in the United States only in the lowlands adjacent to the Pacific and the Gulf of Mexico. Considerable loss among sheep has been reported from Oregon, Montana, California, Texas, Louisiana and Florida. In an abattoir at Logan, Utah, 37 per cent of livers were condemned from cattle in an area where fluke disease was enzootic.[2] Slaughter records for 10 years under Federal meat inspection, obtained from six Gulf Coast packing houses in Texas, Louisiana, and Florida, showed 37.5 per cent of the native cattle and 6 per cent of the calves to be infected with the liver fluke—Olsen.[3] It is widely prevalent in England and Europe, but in recent years the losses have been controlled. It may affect all domestic animals, rabbits, guinea pigs, and man, but it is of chief importance as an enzootic in sheep, goats, and cattle.

Etiology.—The name *Distomiasis* comes from Distoma, a general term including the various genera of trematodes or flukes—parasitic platyhelminths with leaf-shaped bodies. *Fasciola hepatica* (Distomum hepaticum), the most common liver fluke, is 20-30 mm. long by 10-20 wide. At the anterior end is a sucker, and just behind this a ventral sucker, whence the Greek word *Distoma*—two + mouth. Adult and young flukes are found chiefly in the liver and bile ducts, occasionally in the peritoneal cavity and the lungs. The eggs are oval, brown or greenish yellow, 130-145 microns long, and provided with an operculum

at one end. They pass out with the feces and may survive the winter. On reaching water they release a ciliated larva, *miracidium*, that swims and penetrates the snail host within a few hours; after a time it emerges as a motile small fluke—*cercaria*. This attaches to grass, becomes encysted, and appears as a small point about the size of a pin-head. It is now resistant and infectious; upon being eaten the cystic wall is dissolved in the stomach, the cercariae bore through the intestinal walls into the peritoneal cavity, perforate the capsule of the liver and reach maturity in the bile ducts. A few perforate the diaphragm and enter the lungs. Injury results from inflammation and mechanical destruction caused by migration of the flukes. Secondary pyogenic infection sometimes leads to hepatic abscess. They may enter the circulation and even be carried to the fetus, causing infection of the newborn. Infection takes place in the summer and the adults migrate from the bile ducts the following spring. Fluke disease is indirectly responsible for black disease in sheep.

Fasciola magna, the large American fluke, may reach a length of 4 inches. It is said to be most prevalent along the Gulf of Mexico, chiefly to infect cattle, and to cause little harm.

The lancet fluke *(Dicrocoelium dendriticum)* in cattle and sheep has been observed by Baker[4] in several counties in central New York.

Morbid Anatomy.—In intense acute infection, the liver is swollen and congested, the serosa is sprinkled with hemorrhages and sometimes covered with fibrin. Sharply circumscribed small holes may open on the surface; under pressure these exude semi-fluid destroyed liver tissue and young flukes. In less acute types the liver is soft and rough and may show channels under the capsule. Thickened bile ducts are marked by ridges on the surface; when slit, the dark, leaf-like motile fluke becomes visible and may be washed out in great numbers.

In the chronic form in cattle the liver becomes grayish and very hard because of the scar tissue; the bile ducts are greatly thickened and often filled with deposits of calcium phosphate; these may occlude the ducts, and they commonly result in profuse diarrhea.

Symptoms.—The main attack against lambs and young goats usually begins in the summer and fall and becomes advanced in the early winter. Four stages of the disease, as described by Neumann,[5] have been recognized, and lesions common to these periods have been noted in sheep in Oregon by Shaw and Simms.[6] The *first stage, "period of immigration,"* occurs when young flukes first invade the liver. In the cases mentioned by Shaw and Simms the "sheep were dying without showing any symptoms; only in some instances were lesions gross enough to indicate the cause of trouble." Some of the parasites found

were less than 1 mm. in length, indicating an age of not more than ten days.

The *second and third stages* follow a month or two later, when the parasite is mature, the lesions are marked, and the symptoms distinctive; this is the usual form of fluke disease. At first the sheep may be dull, weak, and have pale mucosae, yet the appetite and condition remain good—"*period of anemia.*" Gradually there develop anorexia, irregular temperature, pot-belly, puffy conjunctivae, poor condition, dry wool, submaxillary edema, and finally diarrhea. Simms[6] reports that diarrhea is not a constant symptom, "as many badly infected flocks have been observed which were not scouring." This group of symptoms persists for about two months, "*period of wasting,*" when many of the victims have either died or been slaughtered. Some improve, and finally pass off the mature fluke in the feces during the "*period of emigration*" in the spring. Eggs are abundant in the feces. Swanson and Hopper[7] have described a method of examination of bovine fecal material for liver fluke ova in which feces are strained through 20, 40, and 60 mesh sieves.

Damage results from lower prices paid for heavily infected cattle, 2 to 2½ per cent loss of weight in grown cattle, a death rate of 1 to 3 per cent, reduction of milk flow of 16 per cent, pasture capacity lowered 85 per cent, gains of infected cattle in feed lots about half as much as those of comparable non-infected ones on the same rations, volume of milk in dairy cows reduced, greater loss in the total cattle population caused by subclinical fascioliasis than in the more severe and fatal form in sheep from the same region, and curtailed breeding in animals suffering from clinical fascioliasis.—Olsen.[8]

Treatment.—Carbon tetrachloride (1 cc.) in a capsule has been reported to be highly effective by Shaw and Simms[6], and Montgomerie.[9] This treatment is repeated three times at intervals of three or four weeks. Hexachlorethane is reported by Olsen[10] to be highly effective in the expulsion of liver flukes from cattle and sheep. A hexachlorethane-bentonite suspension is prepared as an aqueous drench by mixing 500 Gm. of commercial hexachloroethane ground finer than 60-mesh size, 50 Gm. bentonite, ¼ to ½ teaspoonful white flour, and 750 cc. of tap water. The dose is 30 cc. for sheep and 20 cc. per 100 lb. of live weight for cattle. Of 110 infected sheep, 104 did not show eggs of the liver fluke in the feces following a single treatment. of 209 infected cattle, 191 showed no liver fluke eggs when examined two or three weeks later. Fasting before and after treatment was not necessary. Calves under 3 months of age need not be treated. While this suspension is reported to have a wide margin of safety, extremely weak animals

should be treated with caution. One dose is usually sufficient to kill the adult flukes in the bile ducts. When the poor or unthrifty condition of the cattle is due to liver flukes, there is generally a remarkable improvement in the weight and appearance of animals within a short time after treatment. A program of drenching cattle in the fall and spring will not eradicate liver flukes, but it will greatly reduce their numbers and improve the health of the cattle. The use of Distol, kamala, and hexachlorethane in the treatment of fluke disease in cows in the Hawaiian Islands has been described by Alicata.[11] A method of prophylaxis by destruction of the snails with copper sulfate has been described by Batte et al.[12]

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DISEASES OF THE URINARY SYSTEM

ANOMALIES OF THE URINARY SECRETION

Anuria

Total suppression of the urine is seen in obstruction of the urethra from the presence of calculi; this is most apt to occur in male ruminants, cattle and sheep, owing to the peculiar anatomy of the urethra. The condition is infrequent, and for this reason it may be overlooked until the animal comes to autopsy. Partial suppression, scanty urination, occurs in acute fevers, acute poisoning by metals or turpentine, and in other severe toxic conditions.

HEMOGLOBINURIA

Hemoglobinuria may result from various causes. In our ambulatory clinic, it is almost pathognomonic of pyelonephritis, this being by far the most frequent cause of blood-stained urine in cattle. In certain years, either hemoglobinuria or hematuria is observed in cows suffering from acute affections of the digestive tract, such as bowel catarrh or enteritis; these cases have been chiefly observed in the fall in cows at pasture. For a considerable time the cause of this form of bloody or blood-tinged urine was obscure. Finally, a definite case of hematuria with blood clots in the urine came to autopsy, when enteritis with apparently secondary nephritis was found. In another case a sudden onset of severe traumatic gastritis was marked by the presence of blood clots in the urine. It is evident, therefore, that in cattle a severe sudden disturbance of the digestive tract may cause hematuria as well as hemoglobinuria. Hemoglobinuria is an occasional symptom in hemorrhagic septicemia, and it is characteristic of bacillary hemoglobinuria and puerperal hemoglobinemia. Hematuria of unknown cause is occasional in young calves; it is also a characteristic symptom of poisoning with copper or mercury or the sulfonamides; it has been observed in septic metritis and mastitis, as well as in sweet clover disease and pyelonephritis.

In sections where piroplasmosis prevails, hematuria from this cause is common. Thus it is not infrequent in Europe, in the tick-fever areas of the United States, and in other parts of the world.

European writers report that the feeding of beet tops and turnip tops to cows may cause hemoglobinuria, but apparently no similar observation has been made in this country.

In azoturia in the horse, hemoglobinuria is one of the distinctive symptoms. It may also occur in the horse in severe impactions of the terminal portion of the great colon. Various conditions causing rapid hemolysis may result in hemoglobinuria. It is one of the symptoms of severe leptospirosis, and of overdosage with certain members of the sulfonamides.

IDIOPATHIC HEMOGLOBINURIA

The name *Idiopathic hemoglobinuria* was applied by Smith[1] of Stillwater, Oklahoma, to a new disease of unknown cause affecting beef cattle, particularly calves, in which the most consistent and readily recognized symptom was hemoglobinuria; apparently age, breed, sex, condition, or feed were not etiologic factors. Similar reports have come from Kansas,[2] Arizona,[3] and Illinois.[4] All attempts to determine the cause, or to transmit the disease experimentally have failed. In Oklahoma it was reported from all parts of the State early in 1942 following heavy fall rains. In the Southwest it appeared in cattle in two or more weeks after they had been shipped from ranges into valley pastures of alfalfa, barley, and oats. In the West Central States outbreaks have occurred on pasture, in feed lots, and even in young calves in box stalls.[2]

Morbid Anatomy.—The macroscopic changes are said to be dependent on the amount and rapidity of hemolysis,[3] thus one writes that the tissues are extremely pale,[4] and another that the cadaver is icteric.[3] There is general agreement that the gall bladder is filled with bile, and that the urinary bladder contains colored urine. One states that subserous hemorrhages are rare,[2] and another that the pleura and peritoneum usually have extensive hemorrhages varying from petechiae to confluence.[3] Other observations are congestion and icterus of the lungs, a liver that is brownish yellow or normal on the external surface, and reddish fluid in the body cavities. The liver is friable and icteric and on section shows extensive necrosis. The disease is said by Roderick[2] to be primarily a hemoglobinemia in that the red cells undergo a marked hemolysis in the blood vascular system with a consequent flooding of the liberated hemoglobin over into the urine.

Symptoms.—The disease is characterized by sudden onset, depression, rapid course, and fatal termination. The temperature ranges from 103 to 105°F., and may drop as the symptoms progress. "The passage of red-stained urine is always the first, and in our opinion the cardinal symptom. In male calves the hair around the prepuce is tinted red, and if the hair on the legs is white it is stained with red urine. Urination does not appear to be painful, but it is frequent. The eyes are

glassy, usually sunken, and the conjunctiva is hemorrhagic and icteric. The animal appears to collapse in a coma."[1] The bowel movements are scanty and hard and the feces may be covered with mucus that is yellow and bloody. The course is from a few hours in calves to three or four days in older animals. The mortality has been reported from 90 to 100 per cent, and the morbidity from 5 to 35 per cent, in Kansas it was less than 1 per cent.

Blood examination reveals a rapid destruction of the red cells. The hemoglobin varies from 20-50 per cent Tallquist, and the red cell count drops from 8 million to 2 million a few hours before death. In one outbreak material sent to Dr. K. F. Myer of the Hooper Foundation showed Leptospira surrounding the tubules of the kidney, but extensive agglutination studies were negative against three strains of Leptospira.

Treatment.—Roderick[2] reports that in the veterinary clinic at Kansas State College, a transfusion of blood from a normal animal is effective in saving approximately 75 per cent of the cases. In Arizona a change of feed to dry hay was followed by recovery of a large percentage of the sick. In Illinois, nine of the ten affected animals treated with sulfamerazine (480 grains) orally, and penicillin (1 million units) intramuscularly, recovered.

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CYSTIC HEMATURIA

(Enzootic Bovine Hematuria; Hematuria Vesicalis)

Cystic hematuria is a chronic afebrile cystitis of cattle of unknown cause characterized by hemorrhagic areas in the mucous membranes, pedunculated tumors, and fibrotic thickening of the wall of the bladder. In America the disease is confined largely to the coastal region of British Columbia, Washington, and Oregon (Kalkus,[1] Hadwen[2]). In Australia an apparently identical form has been described by Bull[3] and associates and it has also been reported from France, Sweden, and Norway. It affects animals of both sexes. The etiology is unknown, but it is definitely not caused by infection and it is not transmissible. It is

irregularly endemic on certain poor farms, only a few animals at a time being affected.

On postmortem examination the internal organs are somewhat pale, and the bladder may be dark because of its bloody contents. Lesions are confined to the mucous membrane of the bladder; early damage results in hemorrhage and mild inflammation of the mucosa; later, vascular channels form in the hemorrhagic areas and finally develop to form hemangiomata—Plummer.[4]

The *symptoms* begin with frequent scanty urination accompanied by straining and switching of the tail. Kalkus[1] states that he has never seen the condition in a heifer previous to calving. The urine may be blood-tinged or uniformly red, and it always contains red cells, even when the color is normal. At first there are no general symptoms, the appetite and milk-flow remaining normal. The initial attack is over a period of a few days to a few months. Following a subsequent parturition there is usually a recurrent attack of increased severity. Emaciation appears, hemorrhage is increased, the mucous membranes are pale and eventually the animal dies or becomes worthless. No effective treatment has been found, but it is claimed that the disease may be prevented by removal from the farm for a period of two months each year.

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ACUTE NEPHRITIS

Stevens[1] writes that the study of nephritis is especially complex "because in the majority of cases we are still ignorant of the precise causes of the various renal changes; because the appearance of the kidneys at autopsy represents only one stage of a process that must necessarily produce according to its duration a great variety of pathologic pictures, and because up to the present it has been impossible to correlate, except in a very general way, the functional disturbances with the anatomical findings." Diffuse non-suppurative inflammatory or degenerative changes of both kidneys have been known as *Bright's disease*, after the description by Bright in 1827. In a discussion by Nieberle,[2] there is presented a classification in which all degenerative changes, such as cloudy swelling of the kidney epithelium, hyaline degeneration, pathological

fatty degeneration, amyloid degeneration, and necrosis resulting from metallic poisons, such as mercury, are termed *nephrosis*, as distinguished from inflammation; while inflammation of the kidney, or *nephritis*, is defined as a derangement characterized by exudation of cellular and fluid constituents of the blood and by tissue proliferation. He also adds that all recent clinical literature shows an intimate relationship between nephritis and certain infectious diseases, such as scarlet fever, angina, and articular rheumatism.

This brief discussion of the classification of nephritis in man is given because veterinary authors have followed such classifications in describing nephritis in farm animals. The various pathological conditions described under Bright's disease constitute an important clinical group in man. The disease is sufficiently separated from its primary affections to present a distinct clinical syndrome and an independent pathology. In domestic animals (herbivora and swine) these conditions rarely, if ever, occur. Diffuse acute inflammations of the kidneys in these animals are practically always incidental to, and associated with, some severe toxic or infectious disease.

Etiology.—The great majority of cases occur in association with the acute infectious diseases, such as anthrax, hog cholera, equine pneumonia, and other acute septic conditions. Frequently nephritis is present as a secondary condition in azoturia. It is induced by such metallic poisons as arsenic, mercury, and phosphorus, as well as by coal tar products, phenol, turpentine, sulfonamides, and various other chemical poisons. While cold and traumatism are recorded as rare causes of nephritis, it is doubtful if they ever cause the disease.

Morbid Anatomy.—Mild forms present no evident gross changes. In more severe types the kidney is swollen, dark, and congested. Hemorrhage is not infrequent, especially in hog cholera. In some cases the kidney is pale or grayish red in color.

Symptoms.—There are no distinctive physical signs of acute nephritis, as in man. The condition may be suspected or recognized because of its known association with certain diseases. A positive diagnosis may be made by means of an examination of the urine. One finds a marked increase in albumin, kidney epithelium, and urinary cylinders. Often there are leucocytes and red blood cells. Occasionally one may find considerable blood (hematuria).

Treatment is directed towards the primary affection.

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CHRONIC NEPHRITIS

Chronic nephritis is a rare disease in herbivorous animals and swine. Nieberle has described three cases in cows observed in the abattoir in Hamburg, Germany. The first was an old animal in medium condition. The kidneys had a combined weight of 7 pounds. The capsule was smooth and easily removed and the underlying surface of the kidney was grayish brown. On section, the cortex was greatly thickened, sharply defined from the brownish-red pyramids, and covered with numerous radiating yellowish streaks. There were many cysts filled with clear fluid. The consistency was normal. The pelvis and medullary substances appeared normal. The second was an old emaciated cow. Both kidneys were greatly enlarged and firm. The Malpighian bodies stood out clearly as fine, gray nodules. The third was an old emaciated cow. One kidney alone weighed 6 pounds. The surface of the kidney was brown to gray, covered with many small, yellowish opaque spots, and finely granular. The consistency was hard, cutting like firm connective tissue. There was a marked thickening of the cortex. As a result of histological examination, Nieberle concluded that such cases, named in the literature as chronic parenchymatous nephritis or "large white kidney," were genuine diffuse glomerulonephritis, and identical with the subacute or chronic glomerulonephritis of man.

The first description that I have found of a clinical case of this disease was that of an 8-year-old mare reported by Fincher and Olafson.[1] At the onset there were swellings about the lips, ears, between the mandibles and beneath the chest which resembled those of urticaria. Under exercise there developed an inspiratory dyspnea, a fast irregular pulse, and heart sounds which could be heard for a distance of several feet. Seven months later the mare had become emaciated, weak, and the heart became irregular and fast under slight movement; death soon followed. Autopsy revealed slight fibrous pleuritis, chronic focal myocarditis, and chronic diffuse glomerulonephritis. A second case, showing similar symptoms and course, has been described by Frank and Dunlap.[2]

That the disease is complex is revealed by statements from Osler that "a clinical classification of chronic nephritis offers many difficulties. A pathological classification deals with end results and cannot be applied at the bedside."

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ABSCESS OF THE KIDNEY

(Embolic Nephritis)

Etiology.—Purulent nephritis is caused by pyogenic bacteria which may reach the kidney in the blood stream, as from a case of traumatic gastritis (Fig. 37), or through the urachus and umbilical arteries, as in navel-ill. It is comparatively infrequent, is seen chiefly in cattle, and is

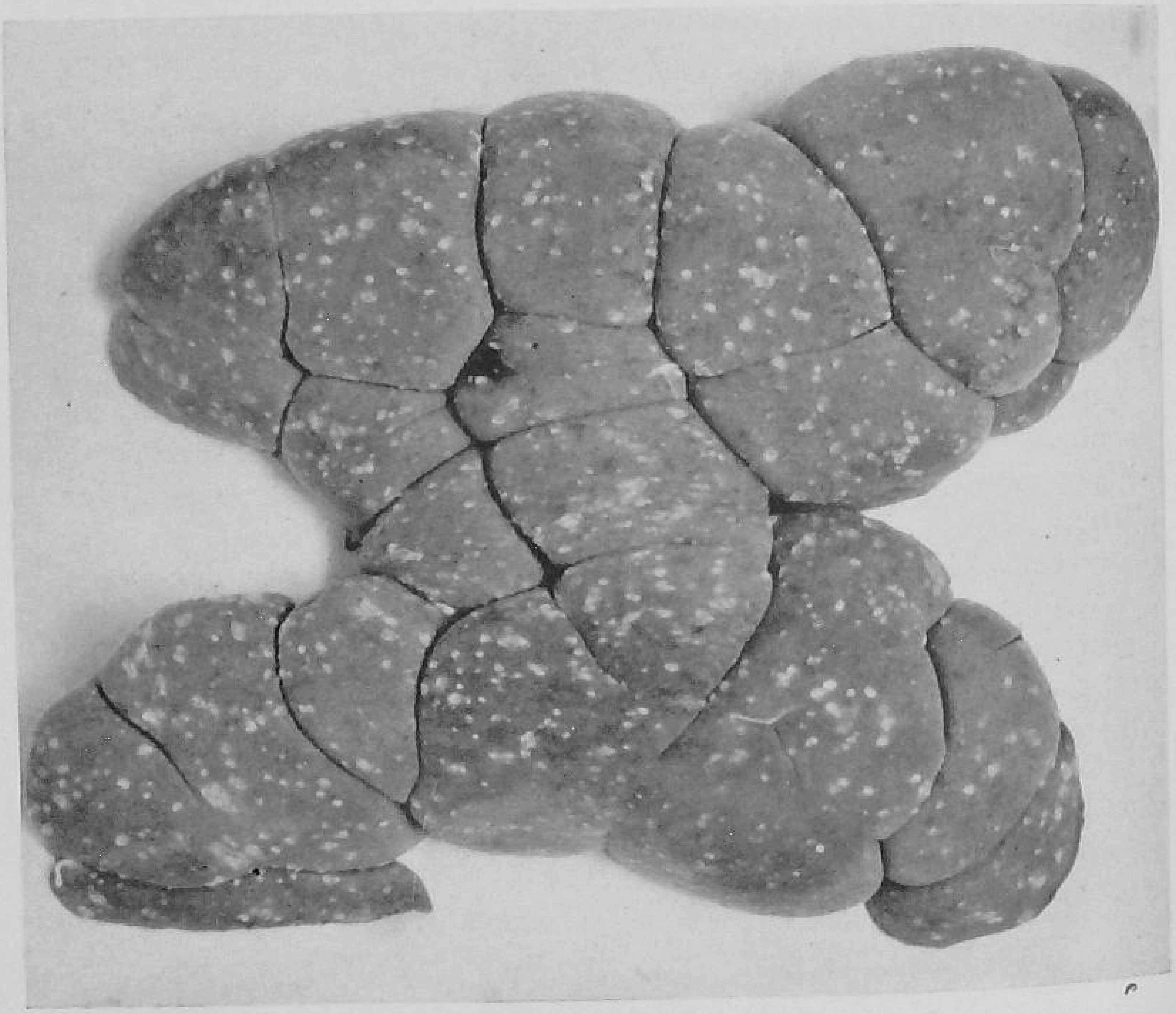

Fig. 37.—Abscess of the kidney secondary to traumatic gastritis in a cow.

always pyemic. The kidney shown in Fig. 37 was from a case of traumatic gastritis with multiple abscess in the lungs as well as the kidneys; cultures yielded *Corynebacterium pyogenes*. Abscess of the kidney is not rare in calves where it results from navel infection. Christiansen[1] has described sixteen cases, fifteen of which yielded *B. coli*. In a 2-month-old calf described by Fincher,[2] the entire right kidney was obliterated by a large abscess. Thus there may be a single large abscess or multiple foci of suppuration. The disease may result from puerperal infection in cows, and from wound infection or strangles in horses.

Fröhner writes that it may be enzootic in swine. It is this form of kidney disease that is caused by *Bact. viscosum equi* in foals.

Symptoms.—The symptoms vary according to the nature of the primary disease. Disseminate purulent nephritis is not recognized except on postmortem examination. In the case of kidney abscess described by Fincher, the calf showed purulent omphalitis, rapid emaciation, and apparent bloat on the right side; trocarization released thin fetid pus containing staphylococci, streptococci, and other bacteria. Extensive kidney abscess might be diagnosed in large animals by means of a rectal examination, or by examination of the urine. There is no remedy.

In kidney abscess in a 15-year-old gelding the animal worked in a normal manner in the forenoon. After dinner he was found down, apparently suffering from posterior paralysis, and not inclined to make any efforts to use the hind limbs. On rectal examination the right ureter was found to be greatly enlarged throughout the entire length, and the right kidney was also enlarged. There was pain on pressure over the lower end of the ureter. Death occurred at the end of four days. The right kidney weighed 10.5 pounds. The capsule was firmly adherent. On section the kidney was found to be filled with pus. The left kidney weighed 3.5 pounds and was congested. In another case in the horse there was a gradual loss in condition over a period of six weeks, with bloody urine for two weeks preceding the fatal termination. An enlarged right kidney was recognized on rectal examination. It is probable that these were cases of pyelonephritis.

References

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2. Fincher, M. G., Omphalophlebitis; abscess of the kidney, Cornell Vet., 1933, 23, 92.

PYELONEPHRITIS

Definition.—Pyelonephritis is a highly fatal, chronic, purulent inflammation of the bladder, the ureters, and the pelvic portions of the kidneys. It occurs chiefly in cows, but has been met with in swine, sheep, horses, and dogs. It is caused by a specific infection, *Corynebacterium renalis.*

While the disease has prevailed in Europe for many years, it was first reported in the United States by Boyd[1] in 1918. In 1888 Hess[2] described the symptoms and lesions, and observed that the disease occurred independent of puerperal infections. In 1891 Höflich[3] described the lesions and isolated the causative organism, giving it the name *Bacillus pyelo-*

nephritides boum. He expressed the opinion that the chief port of entry was through the bladder and not hematogenous. On practically the same date Enderlein[4] published a similar report, naming the causative organism *Bacillus renalis bovis*. Extensive investigations have been reported by Jones and Little[5] of the Rockefeller Institute.

Etiology.—Pyelonephritis in cows has been infrequent in our clinic until 1924. From 1915 to 1924 not a single case was diagnosed. In the eight-year period from July 1924 to June 1932 thirty-four cases were recognized, and 12 of these were in the year 1931-32. Because of the distinctive lesions it is not possible that it had previously escaped recognition. Ninety per cent have been observed in the period from December to May. Apparently the symptoms are aggravated by cold weather. The relation of cold to kidney disease is found in nephritis in man, where it is listed as a cause; according to Osler, however, cold only aggravates an existing nephritis. While the disease is said to occur only in females, Boyd[6] reports that he met with one case in a bull, and the disease in males is also mentioned by McFadyean[7] in a general article on nephritis. There is no special age selection, though the majority are met with at from two to seven years. In 1937 Boyd and Bishop[8] reported the disease in 7 mares and 1 stallion. It is not infrequent in unbred heifers, and Jones has reported one case of spontaneous infection in a 12-day-old calf. Olafson[9] has reported one case in a dog from which *Corynebacterium renalis* was recovered.

A number have regarded the disease as secondary to other pyogenic infections, especially metritis and mastitis, the infection having a hematogenous origin. This view is not now generally accepted for the reason that it is not infrequent in the absence of other diseases, as in unbred heifers. Furthermore, the causative organism is unlike that of other pyogenic diseases, and it has been experimentally reproduced by introduction of pure cultures into the bladder. But when the disease develops shortly after a severe parturient disease, it is reasonable to assume that this condition may have favored the progress of the pyelonephritis. Because of the persistence of pyelonephritis as an endemic affection in certain high-producing and highly fed herds, it has been suggested that a high protein diet may predispose to an attack.

Corynebacterium renalis is a pyogenic gram-positive diphtheroid 2 to 3 microns long by 0.6 wide. Within the body it is found in clumps in smears from the urine or exudate. Studies by Jones and Little[5] have suggested to them that the habitat of the organism is in young calves. They report that the lower urinary tract of male calves may harbor an organism similar in morphology, cultural characteristics, and certain immunological properties to the organism encountered in this

country and Europe in spontaneous infections of the bladder, urethra, and pelvic portions of the kidneys of cows. It seems probable that many cases may really originate as infections during early life. They introduced a culture from steers into the bladders of three cows; this produced a local process which later reached the pelvis of the kidney

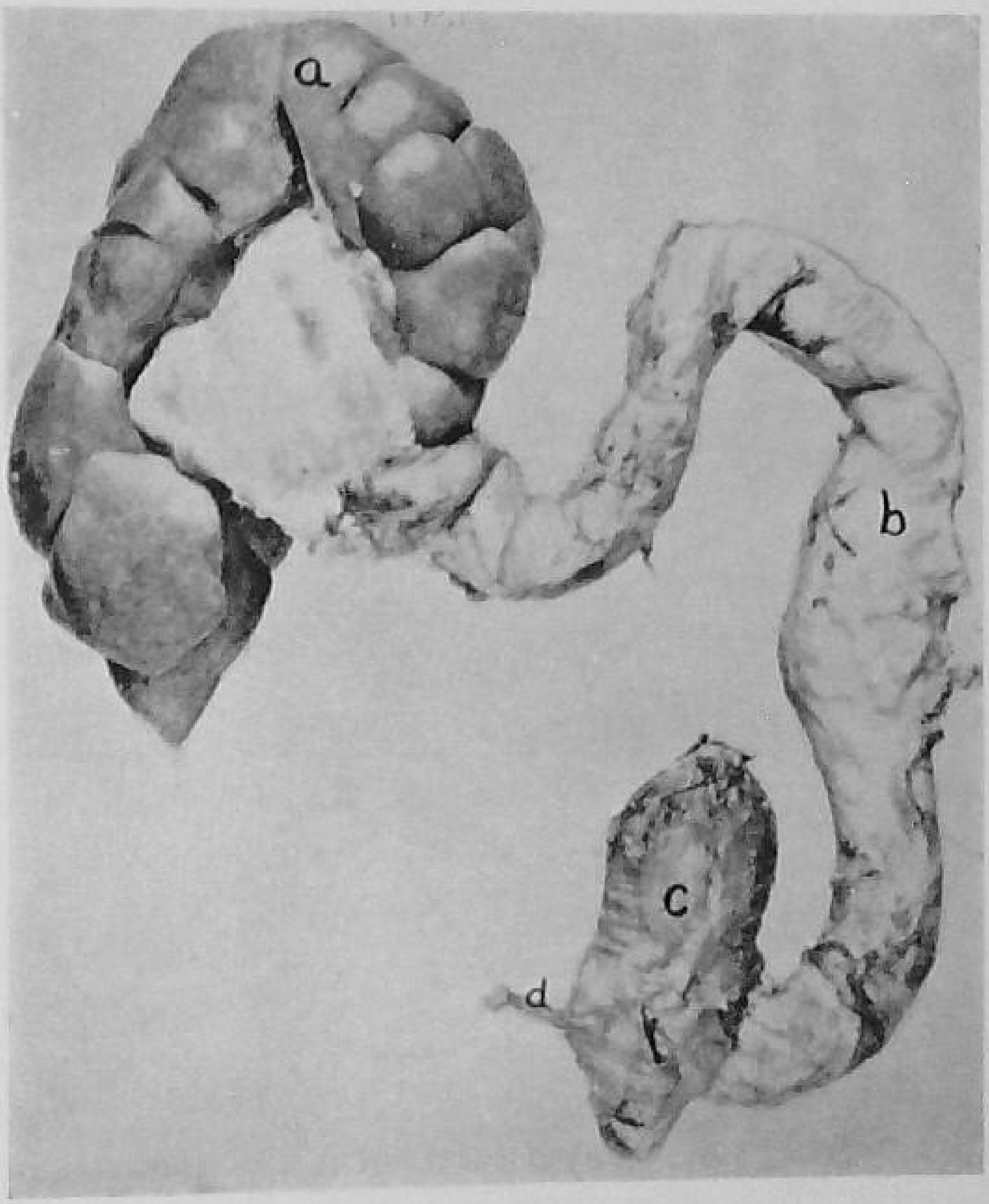

Fig. 38.—Pyelonephritis in cow. Left kidney, ureter, and bladder. Combined weight of organs 12 pounds; a, small renal abscess; b, ureter; c, bladder; d, a short section of the normal ureter. (From Boyd, Cornell Veterinarian, 1927, 17, 45).

by way of the ureter in one. Wester[10] reports producing the disease experimentally by the introduction of sterile sand and a pure culture of *C. renalis* into the bladder followed by massage. So far as known it inhabits only the urinary tract. The mode of infection is unknown, but there is evidence of transmission by contact.

Morbid Anatomy.—In typical cases that have terminated in natural death or by slaughter at an advanced stage, the postmortem changes are highly characteristic. The cadaver is emaciated. The bladder is

thickened, hemorrhagic, and edematous. The ureters are enlarged to several times their normal diameter and the mucosa is hemorrhagic. The kidneys are greatly enlarged in size and weight. The surface may present grayish opaque areas and the normal lobulation may be diminished so that the surface of the organ is more nearly smooth; the capsule may be adherent. On section, the pelvis contains a mucilaginous grayish or yellowish pus, often mixed with urine and blood. There is more or less destruction of the adjacent tissue, and there may be abscess formation in the lobules. The bladder, ureters, and sometimes the vagina contain a mixture of blood, urine and pus. Erosion of blood vessels may result in massive blood clots, or sudden deaths from hemorrhage. In some cases the lesions are largely confined to the bladder; in others, the visible macroscopic lesions are confined to the kidneys.

Symptoms.—Hess[2] described three clinical groups: (1) the onset is like that of a severe acute indigestion; (2) the symptoms are like those of chronic vaginitis and metritis; and (3) the syndrome is that of cystitis. Others have related the similarity between this disease and traumatic pericarditis. In our experience the onset and general symptoms vary widely, yet the diagnosis is seldom difficult if one really examines the urinary system.

The usual history shows a loss of condition over a period of weeks or months; the appetite may be good but the loss of flesh and milk flow is progressive, and there may be diarrhea. Not infrequently the attack is sudden in an animal in good condition with all of the symptoms of an acute indigestion with colic, or there may be a history of intermittent colic. The cow may be restless, or tread with the hind feet or kick at the belly or stand with the back arched. The first symptom to be observed may be the passage of bloody urine and in our experience this is almost pathognomonic of the disease, but it also occurs in parturient hemoglobinemia. The urine may be positive to the Ross test for acetonuria, and there may also be excitability and other nervous symptoms. In other instances the owner notes unusual frequency of urination, perhaps with straining. Occasionally the initial symptoms may follow a severe metritis or a prolapse of the uterus, and in these cases it may be suspected that the parturient disease is primary. The owner may report that the cow emanates a very fetid odor, similar to that of acute septic metritis. In one of these cases the patient was a 2-year-old unbred heifer found down in the pasture. In another instance a cow died from the supposed results of eating her own placenta, only to reveal advanced pyelonephritis on autopsy.

On examination the general condition is nearly always below normal, and often there is emaciation. The mucous membranes, pulse, respira-

tion, and temperature show no change. In the advanced stages, however, there is a marked increase in the pulse rate and the mucous membranes are pale. Examination of the urinary system may reveal a mixture of pus, blood, and urine in the vagina. Palpation through the walls of the vagina may disclose the enormously enlarged ureters at their point of entrance to the bladder, and they may be palpated through the vagina more readily than through the rectum. If the finger can be

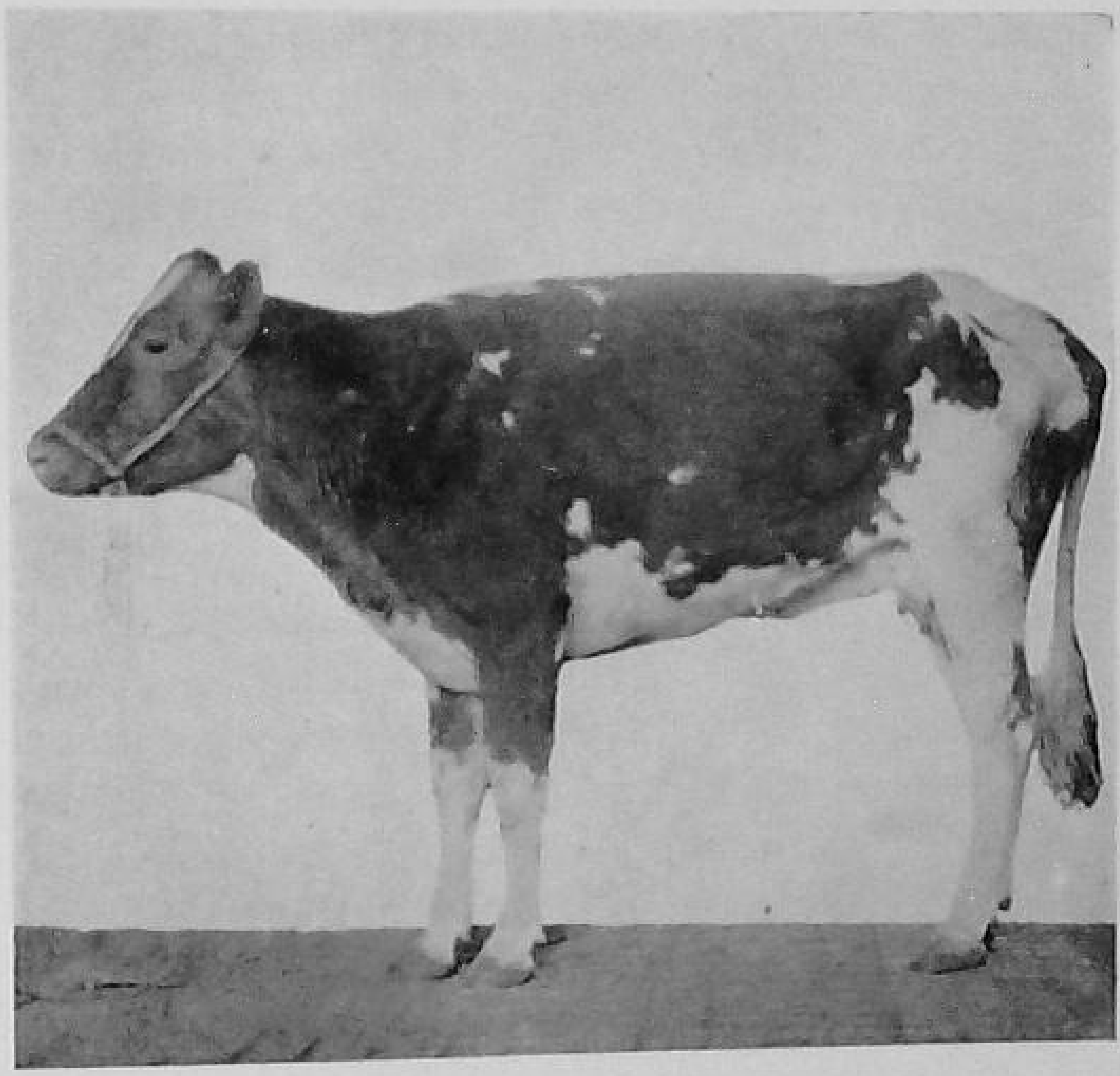

Fig. 39.—A case of pyelonephritis. The attitude and other general symptoms resemble those of traumatic gastritis.

passed through the urethral opening one may recognize the roughened and thickened condition of the mucosa of the bladder. Palpation through the rectum usually reveals a marked increase in the size of the kidneys, and an absence of the normal lobulation; the surface may even be smooth. Pressure on the kidneys may cause pain. The distinct enlargement of the ureters may be detected through the walls of the rectum. There may be periods of improvement, even of apparent recovery, only to be followed by recurrence of the symptoms. Infrequently the kidneys, ureters, and bladder present no abnormal changes recognizable on rectal examination.

In our experience, examination of the urine has shown, without exception, evidence of pyelonephritis. The most constant finding is reddish urine, usually containing blood clots or masses of pus; sometimes the

urine is fetid. Analysis may reveal albumin up to 5 or 6 grams in 1000 cc. With few exceptions, *C. renalis* is found in smears.

The *course* may lead to death within a few days or only after several months to a year. If recoveries occur they are extremely infrequent. A positive *diagnosis* can be made by means of a physical examination of the urinary system, including the urine. While the finding of *C. renalis* is accepted as proof of the nature of the disease, the mere presence of this microorganism may prove to be of less significance. In the differential diagnosis one needs to consider traumatic gastritis, acetonemia, Johne's disease, necrobacillosis of the liver, metritis, and puerperal hemoglobinemia. In one group of young heifer calves showing hemoglobinuria, *C. renalis* was recovered from the urine; there were no other obvious symptoms and no subsequent symptoms of pyelonephritis.

Treatment.—While there is no established effective remedy for pyelonephritis the administration of sodium phosphate, monobasic, (4 ounces, 125 Gm. daily) has been followed by marked improvement in a few cases and it may prove to be highly beneficial. This treatment was first suggested to us in conversation with R. E. Barker of Hereford, England, who reported its action to be beneficial in this disease. It acts as a urinary acidifier and may in this manner combat the effect of the infection which causes a highly alkaline reaction in urine. When administered early in the attack, this method of treatment has been followed by improvement in several cases of pyelonephritis.

A report on the treatment of six cases of pyelonephritis with penicillin has been made by Beck et al.[11] At the time of the report, three cows seemed to have made complete recoveries, hematuria had returned in one and *Corynebacterium renale* had been found in the urine of two. Penicillin procaine, 2 to 3 million units intramuscularly every second day, has proved to be effective when administered early and is now in general use in the treatment of pyelonephritis.

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DISEASES OF THE HEART

Compared with cardiac affections in man, heart disease in animals is rare. The lower animals are seldom affected with degenerative vascular changes, one of the most common causes of heart disease in man; they are less subject to localization of secondary infections in the heart, and they suffer less from physical and psychic strain. The chief causes of cardiac disease in domestic animals are: (1) obstruction of the pulmonary circulation, as in emphysema of the lungs (heaves) in equines; (2) infections and intoxications; and (3) old age, chronic dilatation and hypertrophy in horses. Three chief forms are recognized: pericarditis; myocarditis, including dilatation and hypertrophy; and endocarditis (valvular disease). In addition to these are traumatic pericarditis and myocarditis in bovines, which far exceed in frequency all other forms combined. Congenital anomalies of the heart occur infrequently in domestic animals.

PERICARDITIS

Etiology.—Except in traumatic pericarditis, which is described under the subject of traumatic gastritis, this disease is infrequent. It occurs as a secondary or associated lesion in infections, such as calf septicemia, navel-ill, hemorrhagic septicemia, equine pneumonia, hog cholera, tuberculosis, blackleg, strangles, swine erysipelas, etc. Infection usually enters through the circulation, but it may gain access by continuity, as in pleuropneumonia. Infrequently pericarditis is observed in cows in the absence of a foreign body and of adhesions between the reticulum and diaphragm; in such cases it is probable that a small pointed object, as a wire or pin, has caused a penetrating injury and then been quickly withdrawn. In rare instances pericarditis is observed as an apparently primary condition; in the ambulatory clinic such cases have been seen in a 4-month-old Holstein heifer, a young hog, and a 12-year-old horse.

Morbid Anatomy.—In acute types associated with general infection, the attack involves chiefly the inner serous surface of the pericardium (p. interna). The exudate is serous, serofibrinous, purulent, or hemorrhagic. In one case of recovery in a cow from apparent traumatic pericarditis, subsequent slaughter revealed a reddish, granular surface on the pericardium.

Symptoms.—Secondary acute pericarditis in association with severe general infection is seldom recognized except on autopsy. The circulatory symptoms are a high pulse rate, dyspnea, and venous pulse. In

the absence of pericardial friction sounds a diagnosis is difficult. Chronic tuberculous pericarditis presents a syndrome indistinguishable from that of traumatic pericarditis. In primary acute pericarditis the symptoms appear suddenly in the form of weakness and rapid pulse which soon lead to complete prostration and death. In the case of primary chronic fibrinous pericarditis in a horse described by Stevens[1] the initial symptoms were dullness and constipation. Two weeks later the temperature was 104° F., with rapid loss of weight and strength, weak rapid pulse, mucopurulent nasal discharge, absence of cough, edema of the chest and limbs, and a muffled heart sound. Six weeks later the edema was pronounced and there was a distinct prominence of the jugular pulse. Percussion over the chest revealed pain and an increased area of dullness over the ventral half. The abdomen was gaunt and the feces watery. On autopsy the pericardium was found to be distended with 8 to 10 quarts of yellow thin pus and covered with a thick layer of fibrin.

Treatment is entirely symptomatic, and is like that for cardiac or circulatory weakness in general. Alcohol, camphor, aromatic spirits of ammonia, or caffeine may be helpful.

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ACUTE DILATATION OF THE HEART

(Acute Heart Weakness)

Acute heart weakness occurs in horses of any age from the effects of overwork; it is especially frequent in farm horses in the early warm months in the spring after a long period of idleness. It is also frequent in war horses when forced to continue in transportation lines until they collapse. The *symptoms* are dyspnea, refusal to move, sometimes collapse ("fits"), rapid pulse, distension of the peripheral veins, and sweating. Shortly after an acute attack the pulse is found to be 75 or more, combined with sweating, depression, and inspiratory dyspnea with stertor. Recovery may take place under rest and treatment, or the dilatation may become chronic and incurable. Following an attack the appetite is poor, and exhaustion reappears when the animal is returned to work. The general condition may remain good. In some cases there is a history of a recent acute infection, such as influenza. There may be a nosebleed and dyspnea when worked, as in chronic dilatation. After a time the animal may become anemic and have pale mucosae. Repeated attacks are an indication of chronic dilatation. Sudden death

from circultory failure is not rare, and usually the cause is not determined; in one case reported by Gilyard[1] it was due to a coronary thrombosis.

Treatment.—For relief of the acute symptoms, administer heart stimulants, such as caffeine sodium benzoate 1 to 2 drams (4-8 Gm.), or atropine sulfate 1/4 to 1/2 grain (0.015-0.03 Gm.). A period of rest or light work is desirable.

REFERENCE

1. Gilyard, R. T., Coronary occlusion in a race horse, Bull. U. S. Army Med. Dept., 1944, No. 77, p. 87.

CHRONIC HYPERTROPHY AND DILATATION OF THE HEART

Definition.—This is chiefly an affection of aged horses which causes attacks of collapse and nosebleed, especially when exercised. Pathologically one finds dilatation of the right heart combined with atrophy of the corresponding wall; in some cases there is a total hypertrophy of the heart.

Etiology.—In our experience this condition has been met with occasionally in old horses, and in no other species. The cause is difficult to establish. It seems probable that some are associated with degenerative processes of old age, as in man. Others apparently are sequellae of the acute infections, such as influenza and pneumonia. In one of our cases, in an 8-year-old horse, it was secondary to a well-marked chronic pleuropneumonia. It may result from acute dilatation, or heart weakness.

Morbid Anatomy.—The cardiac lesions are chiefly a dilatation of the right ventricle in association with atrophy of the muscular wall of the right ventricle. In one autopsy in our ambulatory clinic there were areas of complete atrophy of the myocardium of the right ventricle, allowing the pericardium and endocardium to touch. Changes that have been observed in other organs are hemorrhage in the trachea and bronchi, pulmonary infarct, marked pulmonary emphysema, chronic pneumonia, pleuritis, extensive thrombus formation in the branches of the mesenteric artery with multiple abscess of the liver. The heart muscle may be light colored and soft; in some it is undergoing fatty degeneration.

Symptoms.—The usual history is a loss of condition over a period of several weeks, dullness, weakness and sweating when worked. There may be a report of stiffness when moved and of repeated attacks of

bowel catarrh. When exercised the horse may stumble and fall, develop nosebleed, and show extreme inspiratory dyspnea with stertor. Examination reveals pale mucosae, in some cases edema of the subcutis, and a temperature that may be normal or as high as 104° F. Examination of the circulatory system reveals a pounding heart impulse, and with few exceptions a marked increase in the frequency of the beat. Auscultation of the heart may reveal bruits or blending sounds. The pulse is irregular, soft, and often intermittent. There may be a marked jugular pulse. Once this syndrome is well established death occurs within one to three weeks. The horse may drop dead during exercise or die in an attack of "colic" or be found dead with no previous warning.

Treatment consists in giving complete rest and administering small doses of digitalis ½ to 2 drams (2-8 Gm.) daily.

BRISKET DISEASE OF CATTLE

Brisket disease is a hypertrophy of the heart due to the influence of high altitudes. Apparently it has been observed and described only by Glover and Newsom[1] of Colorado. From a study of 45 animals, they demonstrated that the hearts of cattle raised at high altitudes (9000 feet) average 0.879 pounds heavier per 1000 lbs. body weight than the hearts of animals raised at sea level.

The chief *postmortem changes* are emaciation, serous infiltration of the subcutaneous swellings, clear serum in the body cavities, and extensive subperitoneal edema of the intestines and the abdominal wall. There is a hypertrophic sclerosis of the liver and edema of the lungs. The heart is enlarged, dilated, and flabby.

Symptoms.—The disease begins with dullness, inappetence, diarrhea, increased pulse and fast breathing. Calves may die early without having shown any swelling of the brisket. Later, there develops an edematous swelling from the submaxillary region to the brisket, which may also extend to the limbs and the lower part of the abdomen. As the disease progresses the patient shows anemic palpitation of the heart, jugular pulse, coughing, and nosebleed when forced to exercise. The blood shows an increase in hemoglobin and red corpuscles. In fatal attacks the course is from two weeks to three months. Recovery follows removal to lower altitudes.

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MYOCARDITIS

While the terms acute and chronic myocarditis really mean inflammation, they are employed in a broader sense to include degenerative changes. Clinical heart weakness may not depend on recognizable anatomical changes. In animals, myocarditis, as distinguished from dilatation and hypertrophy, is infrequent.

Etiology.—Myocarditis in animals is usually infectious in origin. It occurs in cattle and swine as a fatal termination of foot-and-mouth disease, in stiff lamb disease, in equine pneumonia, in severe septicemia, azoturia, and heat stroke. It may be secondary to endocarditis and pericarditis. Arteriosclerosis, the chief cause of myocarditis in man, is rare in animals. A form of multiple degeneration of the heart muscle in fat swine has been described by Karsten[1] in Hannover. The lesions are like those of foot-and-mouth disease in the form of small, circumscribed grayish or yellow spots uniformly distributed through the muscle. Deaths occur suddenly, and the cause has not been determined. Investigations by Thomas[2] and associates have shown that rats and hogs develop severe myocardial lesions, characterized by necrosis of muscle fibers and marked cellular infiltration when they are fed a diet deficient in potassium and in vitamin B.

Morbid Anatomy.—Three chief anatomical forms are described: (*a*) acute parenchymatous myocarditis, presenting discolored foci, friable consistency, and a light-colored heart muscle ("tiger heart"). Histological section shows degeneration of the muscle fibers. (*b*) chronic interstitial myocarditis with proliferation of the interstitial tissue, and (*c*) cardiac abscess.

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2. Thomas, R. M., Mylon, E., and Winternitz, M. C., Myocardial lesions resulting from dietary deficiency, Yale J. Biol. and Med., 1940, **12**, 345.

ACUTE ENDOCARDITIS

Etiology.—Acute endocarditis is chiefly a metastatic infection from the blood stream caused by various kinds of bacteria. Although it is described as relatively frequent, it has never been observed in our clinic. Puerperal septicemia is the chief cause in cows, and wound infection is said to be the chief cause in solipeds. Secondary acute endocarditis may occur in various infectious diseases, such as swine erysipelas, and equine pneumonia. Apparently there is no distinct line of separation between this form and the far more frequent chronic endocarditis (valvular disease).

Morbid Anatomy.—While two anatomical forms are described, *e. verrucosa* and *e. ulcerosa*, there is no distinct differentiation between the two. With few exceptions the cardiac valves are the seat of the lesions. Verrucose endocarditis is an exudative and small-celled infiltration beneath the endocardium in the form of nodules 1 to 2 millimeters in diameter. These later proliferate to form wart-like polypous growths on the margins of the valves. The valves may also become thickened, and adherent to the ventricular wall. Ulcerative endocarditis is a necrotic inflammation of the endocardium with the formation of various-sized ulcers.

Symptoms.—The symptoms of acute endocarditis are very similar to those of other forms of acute heart disease. There is a marked increase in the impulse and rate of the heart. The pulse is fast, irregular, weak, imperceptible and intermittent. A high fever and dyspnea are usually present. It may terminate quickly in death or continue in an acute form for several weeks or become chronic (valvular disease).

Treatment.—Verrucose endocarditis may recover under the influence of rest and digitalis. Ulcerative endocarditis is incurable.

CHRONIC ENDOCARDITIS

(Chronic Valvular Disease)

Etiology.—Chronic valvular disease is occasional in cows, where it causes symptoms closely resembling those of traumatic pericarditis. While it is said to be the result of acute endocarditis, all of our cases in cows have been chronic without any history of acute heart symptoms, and apparently all have been metastatic from a previous puerperal infection or from traumatic gastritis. In swine, valvular disease is relatively frequent as a sequella of swine erysipelas, and occasionally it may result in a similar manner from hog cholera, and swine plague. In the horse, influenza and pneumonia are the primary causes. Euken[1] has described several cases which were associated with liver-fluke disease in cows. According to Huttenreiter[2] there were 380 cases of valvular disease in horses in the German army in seventeen years. In a 9-month-old calf treated in the ambulatory clinic, cultures from the fibrinous valvular growths yielded *B. coli*, and *Corynebacterium pyogenes* in pure culture was recovered from a sample of blood taken by Fincher from a cow affected with valvular disease of the right heart. In two cows that died of traumatic pericarditis, fibrous growths were found on the left auriculoventricular valves. Kernkamp[3] has reported 19 cases of endocarditis in swine; *E. rhusiopathae* was obtained from 11 and streptococci from 8. They represented about 1 per cent of the

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CHRONIC ENDOCARDITIS

(Chronic Valvular Disease)

Etiology.—Chronic valvular disease is occasional in cows, where it causes symptoms closely resembling those of traumatic pericarditis. While it is said to be the result of acute endocarditis, all of our cases in cows have been chronic without any history of acute heart symptoms, and apparently all have been metastatic from a previous puerperal infection or from traumatic gastritis. In swine, valvular disease is relatively frequent as a sequella of swine erysipelas, and occasionally it may result in a similar manner from hog cholera, and swine plague. In the horse, influenza and pneumonia are the primary causes. Euken[1] has described several cases which were associated with liver-fluke disease in cows. According to Huttenreiter[2] there were 380 cases of valvular disease in horses in the German army in seventeen years. In a 9-month-old calf treated in the ambulatory clinic, cultures from the fibrinous valvular growths yielded *B. coli*, and *Corynebacterium pyogenes* in pure culture was recovered from a sample of blood taken by Fincher from a cow affected with valvular disease of the right heart. In two cows that died of traumatic pericarditis, fibrous growths were found on the left auriculoventricular valves. Kernkamp[3] has reported 19 cases of endocarditis in swine; *E. rhusiopathae* was obtained from 11 and streptococci from 8. They represented about 1 per cent of the

porcine hearts examined in a ten-year period. *Pasteurella suis* was recovered by Sautter et al.[4] from lesions of endocarditis in a 350-lb. sow.

Morbid Anatomy.—The most distinctive lesions consist of cauliflower-like fibrinous growths on the right auriculoventricular valves, but in some instances they may be located on the left auriculoventricular valves.[5] In one cow affected with metritis and impending early abortion, a cauliflower-like extensive growth was an inch thick and 4

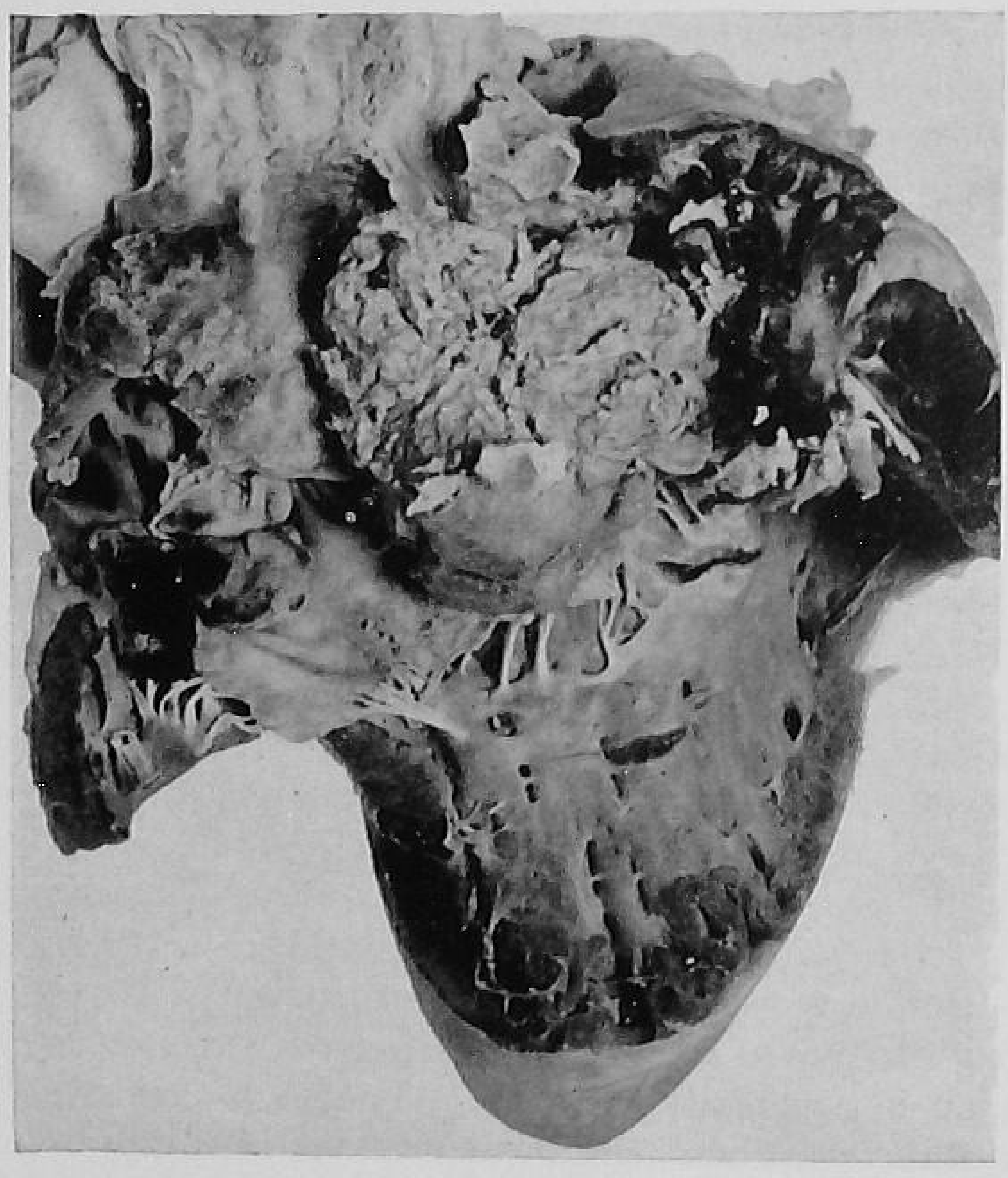

Fig. 40.—Chronic endocarditis, cow.

inches long; it extended from the valves of the right heart to the opening of the pulmonary artery, the valves of which were ulcerative and nodular. The growths are usually about 1 to 2 inches in diameter, yellowish, and soft. They have the appearance of a hematoma in the early stages of organization. Other associated lesions in our cases were: cirrhosis of the liver and ulcers on the abomasum; metritis with impending early abortion; thrombosis of the pulmonary arteries; multiple exudative arthritis; hypertrophy, dilatation, and degeneration of the heart, congestion of the lungs, reddish serum in the body cavities, and chronic

circumscribed traumatic peritonitis. Infarcts and necrosis in the lungs and infarcts in the kidneys are not infrequent.

Symptoms.—The onset may be gradual over a course of several months, as in the case described by Dr. Pickens,[6] or the symptoms may become distinctive within a period of about a week. In all cases in cattle, of which we have records, the ages were from 9 months to 6 years, with the exception of two aged animals in which the lesions were associated with traumatic gastritis. Usually there is a history of loss in condition, appetite, and milk flow. Often the cow has been dull, and there may have been shifting lameness. On examination there is a high pulse rate, as in traumatic pericarditis, from 75 to 150, and there may be a distinct jugular pulse. The poor condition, stiffness, and soreness on percussion over the region of the heart suggest traumatic pericarditis, but no splashing sounds are recognized. The temperature may be normal or as high as 104° F. On auscultation one may hear a muffled sound over one side of the heart and a clear sound on the other; there may be a distinct swish, or the sound may be normal. The course is from a few days to a few months, usually ending in death. Death may occur suddenly and unexpectedly without previous marked signs. Towards the end, dyspnea, weakness, and edematous swellings of the limbs, chest and neck appear. There is no cure.

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DISEASES OF THE BLOOD-FORMING ORGANS

ANEMIA

Anemia is a deficiency of the total volume of blood (*oligemia*), or of red blood corpuscles (*oligocythemia*), or of hemoglobin. It may be due to failure of manufacture in the red marrow of bones, increased blood destruction, or to a loss of blood from hemorrhage. All anemias are secondary; when the cause is unknown the disease may be classed as primary.

Posthemorrhagic anemia may be acute or chronic. The amount of blood that may be lost without causing death varies widely. Cows are relatively susceptible to hemorrhage. It is estimated that 5.5 per cent of the body weight of blood may be lost without causing death, but this is an unsafe guide to follow. Cattle may die within a few hours to two or three days from constant bleeding from a small vessel. This has been observed after expulsion of a corpus luteum from the ovary, after dehorning, following operations on carpal tumors, etc. The theory is often advanced that bleeding from small vessels will cease spontaneously after blood pressure has been reduced from loss of blood, and that this automatic control will become operative before the life of the patient is threatened. Fortunately this is often true, but there are frequent exceptions among bovines. Sudden deaths from spontaneous hemorrhage are infrequent in animals. They may result from erosion of an artery, from a strangles or glanders erosion ulcer in the nasal passages or guttural pouches, from an erosion of a blood vessel in pyelonephritis, pulmonary tuberculosis, or from pulmonary abscesses in cows. We have met with two cases of death from spontaneous rupture of the pulmonary vein in the horse. Sweet clover disease seems to be a form of posthemorrhagic anemia.

Anemias due to increased blood destruction form the largest group. They are associated with faulty nutrition, chronic infectious diseases, ascariasis, strongylidosis, stomach-worm disease, parasitism in general, piroplasmosis, parturient hemoglobinuria, etc. The hemolytic agent may be a bacterial toxin, or a parasitic toxin as in ascariasis. The hemolysis of tick fever is caused by piroplasms. In chronic infectious and parasitic diseases anemia may be due to deficient blood formation as a result of inanition, as well as to hemolysis from products of infection.

Anemia due to deficient blood building is observed in chronic wasting diseases; it is of little importance in veterinary medicine.

The symptoms of anemia are depression, emaciation, paleness of the

mucous membranes and skin, and in extreme cases edema. There are also digestive disturbances, and a weak rapid pulse. There is a decrease of from 50 to 75 per cent in the number of the red blood cells and a marked decrease of the hemoglobin. Excessive and rapid hemolysis is manifested by jaundice, and when the destruction is extremely rapid there may be hemoglobinuria.

SWEET CLOVER DISEASE

Definition.—A fatal hemorrhagic disease of cattle caused by feeding damaged sweet clover in the form of silage or hay. This is considered by Roderick and Schalk[1] as an entirely new disease, undescribed previous to 1922, and without parallel in medical literature. Apparently it should be grouped with the posthemorrhagic anemias.

Etiology.—Sweet clover disease has been recognized and described in many parts of the United States and Canada. That the disease is caused by sweet clover was first reported by Schofield,[2] in Canada, in 1924, when James Brown, a practicing veterinarian, recognized that it was caused by the feeding of damaged sweet clover hay. About the same time it was found by Schalk[3] in many herds in North Dakota and was recognized in several widely separated parts of the Middle West. It has since been recognized in many states where sweet clover is fed, including New York. In a herd of pigs fed ground sweet clover hay, 20 of 65 died of hemorrhage after castration (Jen-Sal Journal, Mar.-Apr. 1940).

The age of onset is chiefly below three years, although Roderick and Schalk[1] report it is probable that mature and aged animals will ultimately present the evidence of hemorrhage if the feeding period is continued with a toxic hay. In feeding tests they produced evidence of hemorrhage in 2-year-olds in an average of 47 days, and in yearlings in an average of 15 days. On a similar diet, rabbits died in from 11 to 13 days. At the time of parturition there is danger of death from hemorrhage, to both the dam and the calf, for substances causing hemorrhage gain entrance to the fetal circulation. Any accidental or operative wound, as from dehorning or castration, is almost certain to result in fatal hemorrhage in affected cattle.

Transmission to young cattle is certain if the hay is toxic. While the disease may be produced in sheep, it is of little importance in this species. It is rare in horses. Rabbits are useful in determining the toxicity of suspected hay or silage.

In 1941, Campbell and Link[4] demonstrated that the hemorrhagic agent is dicoumarin which is formed from coumarin in sweet clover spoilage. Because of the large juicy stalks, it is not easy to cure sweet

clover, and the average crop contains some damaged hay. As a rule this damaged hay is not dangerous. Schalk[3] estimates that where the cattle on one farm are attacked there are perhaps fifty farms where the animals remain unaffected, yet consume damaged sweet clover. It is not the extent of the mold or spoilage that determines the toxicity, and the degree of toxicity can be determined only by feeding. Schalk writes that "while some farmers have successfully fed cattle throughout the entire winter season from stacks that were molded into a solid mass, others have experienced trouble in their herds within two to three weeks after beginning to feed some hay and silage that were molded so slightly that the spoilage could hardly be detected." The disease may appear when the hay is fed alone or in conjunction with other suitable food, and an attack does not confer immunity. It is not caused by grazing on sweet clover pasture. Non-bitter sweet clover (*Melilotus dentata*) does not become toxic on being spoiled.

Morbid Anatomy.—Death is caused by hemorrhage, and no other postmortem change is found. Beneath the skin one finds scattered hemorrhages, or extensive masses of blood which correspond to swellings on the body. One may find extensive intermuscular hemorrhage, principally in the gluteal, lumbar, and shoulder regions. Subpleural and subperitoneal effusions are frequent under both the parietal and visceral membranes; these are most extensive over the rumen. Without exception hemorrhages are found in the heart and pericardium; they are also found in the fetus and in the newborn calf. Within the tissues the blood clots, but in the peritoneal cavity it remains in a fluid condition. Hemorrhage is rare in the mucous membranes, and it has not been found in the lungs, or chest cavity. No other disease presents such extensive hemorrhage. The presence of a large blood clot in the mediastinum combined with extensive subserous and subcutaneous hemorrhages has led to a suspicion of anthrax.

The essential change is thought by Roderick[5] to be an injury to the arterial vascular bed from a diffusible chemical substance. That there is any serious injury to the parenchymatous organs is disproved by the prompt recovery following transfusion. The disease is not transmitted by blood transfusion from the sick to the well. Roderick[5] further states that "the absence of hemoglobinuria and icterus and the low icteric index, together with the failure to find deposition of hemosiderin, indicate that this is not a hemolytic disease."

Symptoms.—There is a history in one or more animals of dullness and stiffness with reluctance to move, and there may have been a few deaths. In many cases there is little depression, and seldom is there any evidence of toxic action. Affected animals move with difficulty and extensive swellings may be found on any part of the body or limbs;

particularly in the hip region, along the sides or ventral border of the chest, and on the neck. The swellings contain blood; they fluctuate in the same manner as hematomas, but do not pit on pressure and there is no crepitation. No symptoms develop prior to actual hemorrhage. The mucous membranes are pale, the pulse and respiration normal, the

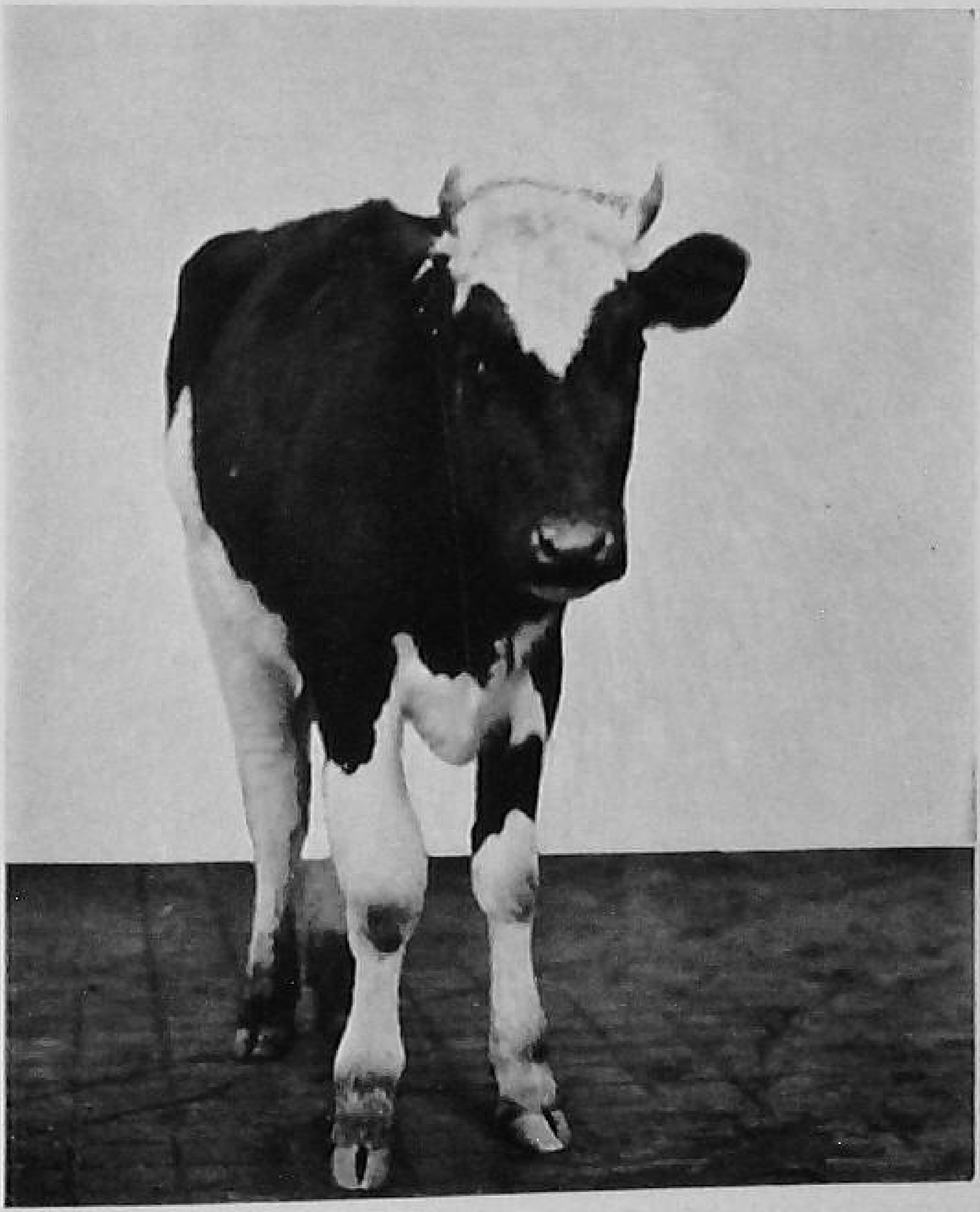

Fig. 41.—Sweet clover disease in a 2-year-old heifer showing enlargement of the right front limb from subcutaneous hemorrhage. Extensive swellings were also present on the left side. Blood transfusion was followed by prompt recovery.

appetite good, and the temperature normal. When hemorrhage becomes advanced there is a marked increase in the rate and impulse of the heart. Epistaxis is not uncommon, but hemorrhage from other mucous membranes is infrequent. Superficial wounds or scratches cause persistent bleeding. As the disease advances, weakness becomes so marked that the animal is unable to stand. Hemorrhages into the central nervous system may cause incoordination of movements and loss of sensation. Cows in late pregnancy abort.

Examination of the blood reveals a loss of from 50 to 75 per cent

of the red blood cells and there is a corresponding decrease in the hemoglobin; these changes are due to escape of the blood from the vessels and not to destruction of the blood itself. There is no decrease of hemoglobin until the appearance of visible evidence of hemorrhage. The decrease in hemoglobin and red cells, and the delayed clotting are the only blood changes that have been recognized. Clotting is determined by drawing 5 to 10 cc. of blood into a test tube having a diameter of 16 mm., and placing it in a water bath at 37° C. The clotting time is determined as the interval which elapses before the blood will remain in the inverted tube. Normally, clotting occurs within ten to twenty-five minutes. In sweet clover disease, the clot may not set for an hour or more, and the time of coagulation always changes previous to the onset of the hemorrhage.

The course is brief and it is especially rapid after parturition. Animals that are apparently normal at night may be found dead in the morning. In the case of slow hemorrhage they may live for a week. In untreated cases the mortality is high.

Treatment.—A change of feed is essential, though additional cases may develop after a week or ten days, the tissues not having had time for repairs. Schalk writes that even after a change of feed the mortality in animals already affected will be 75 per cent or more. Recovery is prompt after intravenous injection of defibrinated blood of normal bovines (500 to 1000 cc.); even after the animal is down this may effect a cure. The serum carries the curative agent. Coagulation is restored in from fifteen to thirty minutes after the injection is completed and recovery is complete in from a week to ten days. In an advanced case in a 2-year-old heifer treated in our clinic with direct blood transfusion, recovery was apparently complete in twelve hours; the owner reported disease in heifers, lambs, and a horse. Defibrination is accomplished by shaking or stirring, after which the blood is strained through gauze; if properly cooled this is effective after several days. Citrated blood is curative in the proportion of 0.3 per cent sodium citrate in a liter of blood.

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PUERPERAL HEMOGLOBINEMIA

(Postparturient Hemoglobinuria; Red Water)

This is a transient rapid hemolysis of well-nourished cows occurring chiefly in stabled animals in the winter and spring two to four weeks postpartum. The essential cause is unknown. It has been prevalent in some sections of Colorado, Utah, and Idaho for a number of years—Farquharson and Smith,[1] and several cases have been diagnosed in New York State. A complete report of the malady as it occurs in Europe has been made by Hjärre[2] of Stockholm; it has been chiefly described from the northern countries: Norway, Sweden, Denmark, and northern Scotland. In Scotland the disease has been recognized for more than a hundred years—Wallace.[3] Its occurrence is chiefly after the third calving at an age of 5 to 8 years when the cows are at the height of production. Usually there is only one case at a time on a farm. While the cause is unknown, it appears to be one of the various disorders of metabolism common to early lactation. The blood plasma of affected cows studied by Madsen and Nielsen[4] was found to be extremely low in inorganic phosphorus, and in 1944 they reported the experimental production of parturient hemoglobinemia in a cow by feeding a ration made up largely of alfalfa hay and dried beet pulp. They concluded that "while all of the factors in connection with the etiology of this disease are not known, this experiment seems to show with reasonable certainty that the characteristic hypophosphatemia was the result of low phosphorus intake and was in some unexplained manner responsible for the erythrocyte destruction." It is not a piroplasmosis nor a puerperal infection. Predisposing influences are poor soil and a preceding dry summer; it is endemic on certain farms, and in Scotland has been associated with heavy feeding of beets and turnips. In Utah and Idaho it is attributed to a diet of alfalfa and sugar-beet pulp—Madsen.[4]

Morbid Anatomy.—The tissues are icteric, and the blood is dark and thin. The liver is somewhat swollen and is yellow or orange-yellow in color due to fatty infiltration and necrosis. Hjärre[2] demonstrated that the liver necrosis is secondary by reproducing it in rabbits by intravenous injections of either hemolyzed serum or hemolyzed blood. There may be swelling of the spleen. The intestines usually contain punctiform hemorrhages, and as a rule the contents of the large bowel are watery.

Symptoms.—The onset is sudden, characterized by hematuria and followed in from two to three days by inappetence and weakness from loss of blood. The extremities are cool. There is rapid loss of flesh, inability to rise, and death in from three to five days. In patients that

recover hematuria is not observed after the first three to six days, and there may be transient hematuria with no other symptoms. Madsen and Nielsen[4] state that "cases which recover seem to show the greatest depression and weakness the first day the urine becomes clear . . . and that the red-cell count usually continued to drop one or two days after the urine had cleared." The urine is either red or dark and often it is positive to tests for acetone. Often there is a transient rise in temperature from 104 to 105° F. The pulse is fast, the heart impulse is increased, and the heart sounds are abnormally loud and sharp—anemic palpitation. The mucous membranes are at first distinctly pale, resembling those of traumatic pericarditis, later they may be icteric. In fatal attacks the red-cell count may drop suddenly from 5 to 6 million to 1.5 million; the hemoglobin drops to 20 to 25 per cent, and there is a marked increase in leucocytes. The bowel evacuations are reported as usually firm and dark, but in the case observed in the ambulatory clinic they were bile-covered, fetid, and watery. Farquharson[1] has observed that convalescents may develop symptoms of pica, such as licking dirt and chewing bones, and the anemic condition may result in necrosis and other vascular changes in the extremities. The course is acute for from three to five days, and convalescence is usually complete in from ten to fourteen days. Hjärre[2] writes that recovery may take from two to three months. Recurrent attacks are not infrequent. The mortality varies from 10 to 40 per cent.

In January a 6-year-old cow that freshened six weeks previously and had shown a subnormal appetite ever since, suddenly refused to eat, the milk-flow dropped from normal, and there was a rapid loss of flesh. Examination revealed emaciation, depression, very pale mucous membranes and a temperature of 104.4° F. The pulse was 70 and the breathing somewhat fast. Rumination was completely suspended and there was severe pain on percussion over the reticulum. The feces were bile-colored, fetid, and watery. On gross examination the blood was dark and watery; the red-cell count was 3 million. Prognosis poor. Administered camphorated oil and dextrose. Four days later there was marked improvement, yet the temperature was 106.5.° Recovery was apparently complete at the end of two weeks. While no hemoglobinuria was observed during the period of treatment, the owner reported that there had been "a red discharge from the vulva."

Diagnosis.—In the beginning this disease resembles pyelonephritis because of the presence of hematuria. In pyelonephritis the course is prolonged, recovery is not the rule, and enlargement of the ureters and kidneys may be recognized on vaginal and rectal examination. Hematuria may also be present in bacillary hemoglobinuria, Texas fever, and in other conditions.

Treatment.—In severe attacks blood transfusion is probably the most effective treatment. Farquharson and Smith advise that the affected animal receive only alfalfa hay with no concentrates and that it be given a powder composed of ferric sulfate and nux vomica, 1 dram each; copper sulphate, 15 grains; and arsenous acid, 3 grains.

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LEUKEMIA

Leukemic Lymphocytoma (Leukemia); Aleukemic Lymphocytoma (Pseudoleukemia)

Definition.—A fatal malignant disease caused by an extensive and abnormal production of immature leucocytes (hyperplasia of the leucoblastic tissue) and characterized by the formation of multiple tumors of the lymph glands; all of the lymph nodes of the body may be enlarged.

While there are variations in the cell structure of these tumors, depending on the origin of the pathologic cells, as from the bone marrow (myelogenous) or the lymph glands (lymphogenous), as well as variations in the appearance of the blood, and in the seat and extent of the tumors, they are classified in one group under the leukemias. According to Feldman's[1] nomenclature there may be an increase in the leucocytes in the blood (leukemic lymphocytoma) or the number may be normal (aleukemic lymphocytoma); either of these may be termed lymphogenous leukemia. Any other form, such as myelogenous leukemia, is extremely rare in domestic animals. It is now commonly believed that at some time during the disease immature white corpuscles appear in the circulating blood. Of forty cases of lymphocytoma in cattle examined by Feldman,[1] the blood was leukemic in twenty-seven. He states that if all cases are considered the lymphoblastomas probably occur with greater frequency than do any of the other neoplasms which affect domestic animals, and that without question it is most common in the bovine species, the horse being rarely affected. Quoting from condemnations for leukemia by the Federal meat inspection service, the ratio in a four-year period was 1:8500 in cattle,

1:149,000 in calves, 1:201,000 in horses, 1:220,000 in swine, and 1:174,000 in sheep. From a study of abattoir material Feldman concludes that "it is among the more common diseases of many of the domestic animals and undoubtedly is the cause of more or less obscure deaths, for unless a thorough necropsy is performed the true nature of the disease may escape detection."

It would be instructive to know the number of condemnations that are clinical cases of leukemia, the number that are condemnations of parts, and the origin of such animals. A survey of the literature reveals only occasional descriptions of the disease. In the reports of the New York State Veterinary College from 1920 to 1940 only two cases are listed in the clinical and autopsy records.

In a period of three years, of 38 cattle that came to autopsy at Manhattan, Kansas, with a record of increase in the blood of atypical lymphocytes, 36 had gross lesions of lymphocytoma—Thompson and Roderick.[2] Information from various sources indicates that this disease is more prevalent in the Middle West than in the eastern part of the United States. Reports of leukemia have been made by Biester and McNutt,[3] Jones,[4] Lund,[5] and Creech and Bunyea.[6]

In certain parts of Germany bovine leukemia has been increasing during the past twenty-five years until it has become a common and important disease. According to Schäper[7] this distribution of the malady has been caused by the use of breeding stock from breeds and families that transmit the disease by inheritance—black lowland cattle of East Prussia.

Etiology.—The cause is unknown. By virtue of unrestricted growth, destruction of normal tissue, and metastasis, its classification by Feldman as a malignant neoplasm is justified. With reference to sex and breed there is no recognizable variation, but there is a suggestion that it is more prevalent in young animals.

Morbid Anatomy.—In animals that have not been slaughtered the cadaver is emaciated. The most important postmortem finding is a symmetrical and marked enlargement of all of the lymph glands of the body. On section, the glands are moist, bulge when cut, are very soft, and may be hemorrhagic in spots. Both the liver and spleen may be greatly enlarged. In the case described by Udall and Olafson[8] the spleen and lymph glands were masses of lymphocytes with little of the normal markings left. In addition to wide involvement of the lymph glands, Feldman also observed extensive metastases and secondary involvement of the lungs, heart, liver, kidneys, and brain; the spleen in many cases was not demonstrably affected. The body cavities may contain large irregular masses, and extensive involvement of the abomasum is frequent. The lesions may be confined to certain areas,

such as the muscle of the heart, the kidney, the abomasum, the spinal cord, or the brain.

Symptoms.—The initial symptoms are swellings of the superficial lymph glands; in cattle these are especially distinct in the submaxillary, prescapular, and precrural regions. At first the appetite remains good and there are no general symptoms; after a few days or weeks there are gradual loss in condition, pale mucous membranes, marked dullness, and labored breathing. Examination reveals distinct enlargement of lymph nodes that are too small to be recognized when normal. Not infrequently there is protrusion of the eyeballs caused by pressure from tumorous masses in the orbit; it was observed in five of a series

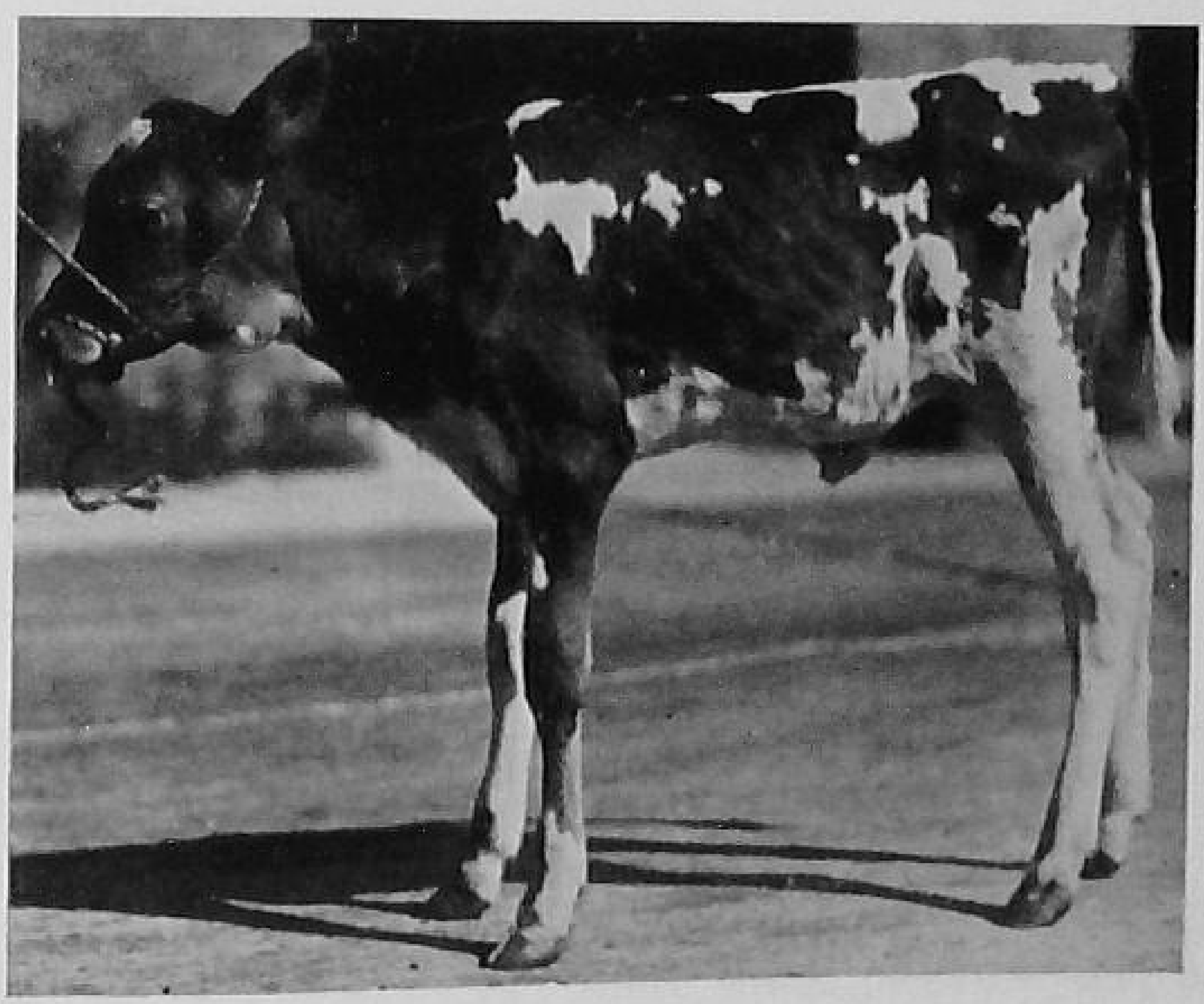

Fig. 42.—Aleukemic lymphocytoma (pseudoleukemia) in a calf.

of forty cases described by Feldman, and in one of the three or four cases observed by the author. Pressure on internal organs from tumorous masses or from single tumors may cause a variety of symptoms, such as indigestion with chronic bloat when the abomasum is affected, posterior paralysis due to pressure from tumorous masses in the pelvis, as well as paralysis and deranged consciousness from a tumor on the brain. The distinctive blood change is a marked increase in the number of white cells; according to Thompson and Roderick, "differential counts approaching or exceeding 65 per cent of lymphocytes should leave little doubt as to the accuracy of a diagnosis of lymphocytosis, and the majority of cases show that as the disease progresses and death approaches the total white cell count decreases gradually from a distinct leucemia to a normal or subnormal level." There is also an

anemia as shown by a reduction in the hemoglobin and the red cells. Death occurs after a few weeks or months.

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ANEMIA IN YOUNG PIGS

(Nutritional Anemia in Suckling Pigs)

Definition.—A highly fatal disease of suckling pigs characterized by a marked decrease in hemoglobin and fatty degeneration of the liver. It is caused by lack of iron and copper salts in pigs confined in indoor pens, on concrete or similar floors, and limited in diet to the milk of the sow.

Etiology.—In recent years there has been a marked increase in losses among young suckling pigs at from three to six weeks of age; this has occurred chiefly in large breeding establishments where improved sanitation against parasitism has been adopted. The seasonal prevalence is chiefly in pigs farrowed in the winter and early spring when they are confined because of the weather, and do not have access to pasture. The blood of newborn pigs usually carries a normal amount of hemoglobin, averaging 10.75 gm. per 100 cc. Observations by Hamilton and associates,[1] and others have shown that "a rapid decrease occurs during the first week to a level but little more than half that at birth, and continues for four to five weeks if the pigs are left indoors," while "removal to an outdoor cindered pen, with no access to soil or vegetation, brings about a prompt recovery, both in blood picture and physical condition."

Doyle[2] has reported that normal hemoglobin is maintained in pigs turned out on blue-grass sod, and also, "that access to blue-grass sod inside of a hog-house protected the pigs against anemia while enabling them to grow at a normal rate."

Hamilton[1] also reported that normal hemoglobin is maintained in indoor pigs, fed exclusively on the sow's milk, kept from the dam's feed and feces, and given daily, by mouth, water solutions of ferric sulfate and copper sulfate in amounts containing 25 mg. of iron and 5 mg. of copper.

Explanation of this deficiency is found in the low blood-forming elements, such as iron and copper, in the milk of the sow. When the newborn pig is limited to milk, and deprived of access to minerals which are available under less artificial conditions, the hemoglobin content of the blood is soon exhausted. From the available evidence, it is apparent that the disease is the result of mineral deficiency, rather than housing, since a supply of sod to indoor pigs prevents the disease. The disease is most prevalent in the modern hog-house, designed to prevent contamination with soil containing parasitic eggs. It is of little or no importance on the dairy farm where only a few swine are kept, and where pigs have access to organic matter.

Morbid Anatomy.—Usually the cadaver is normal in appearance, but in prolonged attacks it may be emaciated. The thoracic and abdominal cavities contain serum and fibrin. The liver presents the most constant gross lesion in the form of marked fatty degeneration; it is slightly enlarged, has a grayish yellow mottled appearance, and often is hemorrhagic. The heart is greatly dilated, even displacing the lungs. The kidneys are pale and affected with subcapsular hemorrhage. The blood and muscles are pale. Pneumonia is frequent. The spleen is enlarged and firm. Recovered pigs may be left with fibrosis of the liver. In the excellent descriptions of this disease by Doyle and coworkers,[3] "hobnail" livers as a result of chronic fibrosis are mentioned. On microscopic examination of the liver, spleen, and bone marrow, active hematopoietic centers are found.

Symptoms.—While the age of onset is usually at about three weeks, some of the pigs may be dull and inactive at as early as one week of age, and anemia has been observed even at birth. Affected pigs may be well-developed and apparently well-nourished, but they show fatigue, dyspnea, thumps, and depression on the slightest exertion. The anemic pigs may be fat, yet the muscles are flabby and the skin and mucous membranes are pale. When about three weeks old, the fat and apparently well-nourished anemic pigs die suddenly. Individuals which survive may become weak, thin, and permanently unthrifty.

The *blood* undergoes a rapid decrease in hemoglobin. In affected groups this starts soon after birth, often at about two weeks, and may continue until concentrations have dropped from the normal of from

9 to 15 grams per 100 cc. to 2 to 4 grams per 100 cc. of blood. The erythrocytes fall to from 3 to 4 millions. Doyle considers as anemic all pigs, three weeks or more of age, which have 3,000,000 or less red blood cells per cu. mm., and 3.8 grams or less hemoglobin per 100 cc. of blood. There seems to be no material deviation from the normal in the white cell count.

The course is chiefly over the period from three to six weeks of age, and the mortality is high. It is estimated that the annual loss in the United States of pigs in the suckling age is about 35 per cent. Much of this is caused by anemia. If the pig survives to the age of approximately six weeks, there is a gradual recovery because of access to foods containing the essential copper and iron.

Prevention and Treatment.—The rapid deterioration of the blood of confined suckling pigs, when allowed only sow's milk, is prevented by the addition to the pig's diet of a small amount of iron and copper. The deficiency is not met by adding iron and copper to the diet of the sow. Hart and coworkers[4] observed that a daily dose of iron sulfate to (25 mg. of iron) and copper sulfate (5 mg. of copper) is sufficient to prevent the anemia. This is provided by giving each pig a solution made of 3.6 ounces iron sulfate dissolved in 5 quarts of water; feed one dram of the solution daily. A more convenient method of administration, and one that causes less shock to pigs that may possibly be anemic, is to paint the udder of the sow daily with the following mixture: 1 pound ferrous sulfate, 2.5 ounces copper sulfate, 1 pound sugar, and 3 quarts of water. A still simpler method of supplying the required iron and copper is to provide newborn pigs with access to grass sod, either in an outdoor yard, or by addition to the indoor pen. While the use of sods in the pen may be successful, it is not always sufficient. Copper and iron sulfate may be added to clean dirt and placed in the pens daily. Schofield and Jones[5] wrote that the most practical method is by the use of a single dose of reduced iron (300 mg.) when the pigs are from 6 to 8 days old. In a group of sows in which the litters were all expelled dead, Archibald and Hancock[6] found anemia in both the aborts and the sows. To the remaining pregnant sows they fed reduced iron (2 Gm.) twice daily for two weeks, and then the same quantity twice a week. At the end of three weeks there was a marked increase in the hemoglobin, and ultimately they gave birth to normal living pigs.

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DISEASES OF THE NERVOUS SYSTEM

INTRODUCTION

The *hemispheres of the cerebrum* are the seat of the psychic functions—thought, feeling, and will, as well as many motor functions. All motor fibers controlled by the will originate here. Derangements of consciousness, therefore, are always referable to this part of the central nervous system.

The *midbrain and cerebellum* are the seat of harmony and equilibrium of movements. The *spinal cord* and *peripheral nerves* function chiefly in the transmission of sensory and motor impulses, and in the control of organs having involuntary musculature; they contain the centers of respiration, of circulation and of all involuntary movements.

Nervous symptoms in domestic animals may conveniently be divided for interpretation into five groups:

(1) *Psychic Derangements.*—Disturbances of consciousness—excitement, depression.

(2) *Motor Irritation.*—Spasms, tremors, muscular twitching, chorea-like movements.

(3) *Disturbances of Motility Proper.*—Paresis or paralysis that may be cerebral, cerebellar, spinal, or peripheral.

(4) *Derangement of the Sensation.*—Hypersensitiveness, hyperesthesia, anesthesia, paresthesia, pruritus, disturbance of the sight, and hearing.

(5) *Disturbances of Coordination.*—Vertigo, ataxia (rare in animals).

Disturbance of Consciousness.—(*a*) *Irritative disturbance* is manifested by excitement, restlessness, delirium, or mania. *Mania* represents the highest degree of disturbance, and is marked by greatly increased impetus to motion. The movements are violent and purposeless, as in rabies and in some cases of enzootic encephalitis in equines. *Delirium* occurs in many of the acute infections (meningitis, meningismus), vegetable intoxications, and poisoning. It is manifested by aimless wanderings, reckless movements, rolling, etc., as in colic. Such wanderings, usually in connection with motor irritation, are sometimes noted in the early stages of milk fever, acute metritis with ketosis, in senechio poisoning, and lead poisoning. *Excitement* occurs most often as the initial symptom of hyperemia, inflammation, or other acute brain disturbance. It is seen in the early stage of milk fever, in malignant head catarrh, and in many other conditions.

(*b*) *Depressed disturbance of consciousness* is characterized by *dullness, sopor* (profound sleep), and *coma* (acute unconsciousness with loss of reflexes).

Both irritative and depressed conditions are always the result of irritation of the cerebrum. Usually they are due to conditions that cause an increase in the intracranial pressure—cerebral congestion and hemorrhage, meningitis, contusions, lightning-stroke, hydrocephalus, encephalitis, abscess, degeneration, tumors (cholesteatoma), lead poisoning, marked degeneration of the liver, bacterial toxins, and parasites (gid).

Motor Irritation.—Motor irritation is caused by an irritative lesion. It is the most common nervous symptom in domestic animals, and spasms and convulsive movements are the most frequent examples. *Clonic spasms* are involuntary quick jerkings of the muscles which follow one another rapidly without interruption, as in trembling—tremors, trembles, involuntary quivering. If the disturbance includes many muscles at one time it is termed a *clonic convulsion*. A *tremor* is a frequent involuntary rhythmical movement. *Fibrillary twitchings* are contractions of single fiber bundles of any given muscle. *Tonic spasms* are prolonged contractions of individual muscles. *Tetanic contractions* are tonic spasms affecting muscle groups, or the entire body; *tetany* belongs in this group. *Forced movements* are examples of motor irritation; they include: (1) *Rotation* around the transverse or longitudinal axis, as observed in gid in sheep, and in lesions in the cerebellum. (2) *Circling* to the right or left, as in lead poisoning, listerellosis, cerebral tumors, or lesions in the cerebellum. Changes in the right side of the cerebrum cause circling to the left, while those in the left side cause circling to the right. (3) *Pressing forward* occurs in encephalitis, toxemia, and cerebral hyperemia—malignant head catarrh, sinusitis after dehorning. (4) *Backing* is observed in hemorrhage at the base of the brain, in purulent meningitis in cows, and in various toxic conditions.

Various forms of motor irritation are met with in a wide variety of conditions; among these are epileptiform attacks in horses, rarely in cattle; absorption of toxic material from the digestive tract—abdominal vertigo; meningo-encephalitis of the newborn; indigestion in cows—occasional; intracranial pressure following injury to the horns; encephalitis; tuberculosis of the brain; pyemic meningitis secondary to metritis in cows; primary diffuse affections of the liver—cirrhosis, acute fatty degeneration in cows; and botulism.

Muscular tremors often precede collapse in acute disease. The term "*Trembles*" is also used to identify certain diseases for which a more definite diagnosis is lacking. One of these is so-called trembles or shivers

in newborn or young pigs. It has been observed in suckling pigs on pasture, when there was failure of the sow to supply milk and the diet consisted exclusively of grass. It may be an initial symptom of hog cholera in suckling pigs. And Kernkamp[1] has described a congenital form of trembles in pigs in which the clonic muscular spasms are invariably present when the pig is born—"*myoclonia congenita.*" Kernkamp writes that it may involve practically all of the voluntary muscles of the body or only certain muscle groups, that the contractures may be mild or marked, and they may be present continually or occur more or less intermittently. In severe attacks there is difficulty in walking and suckling with death from starvation; in milder forms recovery may occur in six weeks.

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Disturbance of Motility Proper (Paralysis, Paresis).—This is caused by destructive lesions in the motor tract affecting the cerebral cortex, the motor pathways, or the muscles, and it is frequent in animals. Paralysis of central origin may not readily indicate its source unless associated with dullness or some other brain symptom. Thus, in infectious encephalitis (Kansas horse disease) there is a marked disturbance of consciousness—mania, sopor, coma as well as paralysis. In botulism, on the other hand, there is no disturbance of consciousness; it is a peripheral paralysis. Central paralysis is caused by inflammation of the brain (Borna disease), tumors, hyperemia, concussion, etc. Often it is the final stage in a series of symptoms beginning with disturbance of consciousness and motor irritation, and terminating in loss of consciousness and paralysis. Central paralysis may also be caused by toxic substances which enter the circulation from diseased organs in other parts of the body, as in metritis, pregnancy disease in sheep, or cirrhosis of the liver, when a correct interpretation may be difficult. Paralysis is of frequent occurrence in cows, as in milk fever, acetonemia, and other conditions which are not well understood. Paralysis with marked tympany has been observed in cows after blows or fracture of the skull that resulted in hemorrhage of the brain.

In general, motor irritation is caused by irritative lesions affecting the motor tract, while paralysis is caused by destructive lesions.

Derangement of the Senses.—Sensory disturbances such as anesthesia, paresthesia, hyperesthesia, loss of sight, hearing, and equilibrium are occasionally of value in making a diagnosis. *Paresthesia*, a sensation of itching or burning, is seen in the horse at the seat of the bite in

the early stages of rabies; in cows, on the fetlocks during the early stages of myelitis, and in pseudorabies; as a transient symptom in some cases of acute septic metritis; in acetonemia, and in spinal paraplegia of unknown cause in bovines. It is manifested by rubbing, licking and biting the part. *Hyperesthesia* is infrequent; in one instance it was due to meningitis in the cervical region in glanders: in another it was distinct in a cow in the first stages of milk fever with acetonemia.

CEREBRAL HYPEREMIA

It has been the custom to apply the term "cerebral hyperemia" to a group of symptoms that are clearly due to an irritation of the brain and from which recovery in a few hours is common. It is not always clear that the condition is a hyperemia. It is possible that in some instances we are dealing with a mild form of meningitis, while in others the syndrome arises from the effect of toxins in the blood that act on the brain yet cause no recognizable lesions—*meningismus*.

Etiology.—Affections causing this syndrome are sunstroke, the first stages of meningitis, and inflammation of parts adjacent to the brain. Thus it occurs in sinusitis in cows after dehorning, in malignant head catarrh in cattle, in rare instances in sheep from a severe invasion of the sinuses by the larvæ of *Oestrus ovis*, and from injuries involving the meninges. Bacterial toxins may cause similar symptoms, as in metritis in cows; in this case one can be fairly certain that the lesion is hyperemia, for when such an attack is fatal extensive meningitis is always present. Intestinal intoxication from spoiled food may account for some of the transient "hyperemic symptoms" not otherwise explained. In this group are also included acetonemia and milk fever.

Symptoms.—Hyperemia of the brain is characterized by disturbances of consciousness in the form of dullness, restlessness, excitement, or vertigo. In association, one commonly notes various degrees of motor irritation, as muscular twitchings, clonic or tonic spasms of the neck muscles, and involuntary movements—pressing the head against the wall, lunging forward in the stall, walking in circles, etc. Paresthesia may also occur, as shown by persistent licking of the feet or other parts. Paralysis of certain parts, as monoplegia or hemiplegia, is absent.

Food intoxication in equines may cause slight vertigo in which the animal stands in a fixed position and staggers when forced to move. It may cause excitement followed by dullness. Other symptoms attributed to the same cause are vertigo, muscular twitchings, epileptiform attacks, involuntary movements, and spasms. In severe attacks there may be general paralysis. The horse may fall when turning, rise

with difficulty, jump in a startled manner when approached in the stall, and recover promptly when a laxative begins to act. In sinusitis after dehorning the cow may stand with the head turned to one side—tonic spasm of the neck muscles, or press forward against the stanchion—forced movement.

On autopsy, animals that have manifested clinical signs of hyperemia of the brain in such affections as lead poisoning and septicemia have revealed active congestion of the meninges.

SUNSTROKE

(Heat Stroke; Thermal Fever; Insolation)

Etiology.—Heat stroke is caused by prolonged exposure to excessive heat, either from the sun, from overexertion in a hot humid atmosphere, or from confinement in a close hot place. Animals fatigued by travel and confined in cars, stockyards, or exposition grounds under a blazing sun with still atmosphere are often victims. Northern animals are said to be more susceptible than those in the South. Lack of water is a predisposing cause. Sheep and heavily haired animals are easy victims of the heat. The cause is due to excessive heat rather than to chemical action of the sun's rays.

Morbid Anatomy.—The blood fails to clot and the veins are greatly distended. The liver and kidneys are degenerated. Rigor mortis and putrefaction develop early. Congestion of the lungs and brain is intense.

Symptoms.—In horses at work the onset may be gradual and manifested by dullness, stumbling, and increased frequency of breathing; the symptoms may not appear until after return to the stable. The skin becomes dry, an important sign of impending stroke. Collapse, followed by restlessness, delirium, convulsions, or coma is frequent in all species. On general examination one observes congested mucosae, palpitation of the heart, fast pulse, hyperpyrexia—106°-110° F., and marked distension of the peripheral veins. The more rapid and irregular the breathing, the more unfavorable the prognosis. The course is always short; some die in from one-half to two hours, others on the third day. The atmospheric condition and the high fever are distinctive. Mild attacks have been confused with indigestion, and pulmonary hyperemia. The death rate is high during a protracted heat wave. With a high temperature and marked prostration or delirium the prognosis is unfavorable.

Treatment.—The chief indication is to reduce the temperature by rapid cooling of the body. This is done by means of cold packs to the head and enemas of cold water, or the body may be sprinkled from a

hose. In one cow, the cold application to the spine induced convulsions, but these were avoided when the ice packs were limited to the head. Shade and free circulation of air are highly desirable. Spasms and convulsions demand narcotics—chloral, chloroform. In coma, bleeding freely from the jugular is reported as useful in man, but it is of doubtful value in animals. Keyser[1] reports favorably on the results of bleeding, followed by intravenous infusion of 4 liters of artificial serum, or the same amount of 0.7 per cent sodium chloride solution. Stimulants may be used freely: caffein sodiosalicylate (4 to 8 Gm.), aromatic spirits of ammonia, strychnine sulfate, or camphorated oil.

In the treatment of numerous cases of heat exhaustion in army maneuvers, often combined with laminitis, chief importance was given by Campbell[2] to intravenous injection of 1000 to 1500 cc. of a 10 per cent saline solution (sodium chloride, 4%; sodium carbonate, 5%; potassium chloride, 0.5%; magnesium chloride, 0.05%). Those that would not eat were given a second intravenous injection after an hour, and a third two hours later. Within 15 minutes after injection the animals would drink gallons of water. For the laminitis the feet were packed in ice, and on the following day and each day thereafter, forced, but gentle, exercise was given.

In a discussion of the prevention and treatment of heat stroke and heat exhaustion among workers on the Hoover dam, Zwalenburg[3] reports that liberal use of water and salt is a useful prophylaxis, while intravenous and subcutaneous injections of salt and dextrose solutions in acute heat exhaustion have given most gratifying results. He writes that "autopsy records show an empty heart and a congested skin after sudden death from heat exhaustion and sunstroke. Evidently heat dilates the capillaries in the skin and allows an outpouring of water from the body until there is not enough fluid left to fill the blood vessels sufficiently to keep up pressure in the brain. The cyanotic purple look of a victim of heat exhaustion has kept observers from seeing that the patient has not enough water (blood) rather than too much and in need of bleeding, as was the custom not so long ago." The improvement which follows sprinkling of animals with cold water is partly explained by its action upon the dilated peripheral vessels.

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LIGHTNING STROKE

Groups of animals assembled under a tree at pasture are often struck by lightning. Oak trees are said to be especially dangerous, but the accident is common in localities where oaks do not grow. According to experimental evidence "death occurs (*a*) as a result of inhibition of the nerve centers, thus bringing about respiratory arrest, while the heart still continues to beat, or (*b*) the heart is also inhibited with the respiratory centers"—Nelson.[1]

Morbid Anatomy.—Often it is difficult or impossible to find post-mortem changes that positively identify lightning stroke. Reeks[2] states that an irregular characteristic line of superficial singeing of the hair is nearly always present; commencing at the tip of one of the ears, it runs down the neck, often in the jugular groove, to one of the fore limbs and so to earth, marked in its course merely by a superficial singing of the hairs; or there may be found a similar line communicating in the region of the croup, and again gaining the earth by way of one of the limbs. Reeks believes that this singeing is present in as high as 90 per cent of deaths from lightning, while others give a much lower figure. Superficial or severe burns may be found on any part of the body or head. Evidence of instant death suggests lightning; the animal may have dropped suddenly while grazing and still hold the last tuft of grass clenched by the incisor teeth. Bloody foam may appear at the mouth and nostrils. Rigor mortis is usually present. The blood is fluid and the spleen and liver congested, as in death from asphyxia. There are no gross changes in the central nervous system. The lungs are congested and may show various degrees of subpleural hemorrhage. As compiled by Hubbard,[3] certain observers attach great importance to contraction of the left ventricle of the heart, but such contraction is often absent in death from electric shock. Severe mechanical injury may result from a heavy stroke.

Symptoms.—Injured animals are left with sensory and motor paralysis affecting chiefly the limbs over which the current passed on its way to the earth. This paralysis is usually transient, and is said never to affect the bladder or rectum, being peripheral in character. Paralytic conditions may be associated with vertigo, blindness, and various degrees of depressed consciousness. Deep mechanical injury may leave the victim permanently crippled, but this condition is relatively infrequent. As described by Maxfield,[4] the symptoms in a Hereford bull 48 hours after an electric storm were recumbency, extreme listlessness, lowered head, drooping ears and eyelids, retardation of reflexes, paralysis of the pelvic quarters involving the rectum and bladder,

with functional paralysis of the digestive organs and muscles of mastication.

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MENINGITIS

(Meningo-encephalitis)

Definition.—The term cerebral meningitis includes inflammation of the dura (pachymeningitis), the pia (leptomeningitis), and to some extent the cerebral cortex. In animals it is the most frequent disease of the brain and it is usually secondary. There are two chief forms: hemorrhagic and purulent.

Etiology.—Meningitis is usually secondary to one of the following causes: (1) *Tuberculosis*, rarely other specific chronic infections. (2) *Pyogenic infections* extending from the cranium (sinusitis after dehorning), navel-ill in lambs and calves, castration wounds, amputation of the tail in lambs (purulent), septic metritis and traumatic gastritis in cows, contusions and fractures of the skull. (3) *Acute infections,* as anthrax, blackleg, hemorrhagic septicemia, hog cholera, septicemia in the newborn, and contagious equine pneumonia. (4) *Poisoning,* especially from lead. (5) *Acute indigestion and enteritis* in cows sometimes ends in meningitis. (6) *Heat stroke,* parasitic larvae, and perhaps some cases of milk fever. (7) *Unknown causes* may operate in either cattle or horses, when one can only surmise that it may be a poison, an alimentary toxemia, or an infection of the brain. Occasionally a death from what appears to be meningitis reveals no definite lesions on autopsy. As a rule, however, when an animal with well-marked signs of meningitis comes to autopsy definite lesions are found.

Morbid Anatomy.—The brain lesions vary widely. Congestion and hemorrhage of the meninges and choroid plexus are the most constant changes. In purulent types, as from navel-ill and wounds, one may find suppurative areas on the cortex; these show polymorphs on smear. Often the cerebrospinal fluid is increased; it may be tinged yellow or red, and is seldom turbid. In general the changes are diffuse, even when the symptoms indicate a focal lesion involving the cranial nerves (ear drop, ptosis, strabismus).

Symptoms.—While individuals vary widely in action, the group manifestations are similar, *motor irritation* being one of the most constant. One sees clonic spasms of the neck muscles, retraction of the

head and neck, twitching of various groups of muscles, and sometimes convulsions. Involuntary movements, such as pressing forward and circling are not rare. Disturbance of the consciousness, as excitement followed by depression, is common, yet complete loss of consciousness is rarely met with in meningitis, and in certain instances there is no apparent disturbance of consciousness. In one 7-month-old Hereford heifer affected with purulent meningitis the chief symptoms were tetanic contractions of the body musculature and attacks against people. Vertigo-like symptoms are occasional; the animal walks with difficulty

Fig. 43.—Encephalitis of the cerebellum in a calf. Circumscribed degeneration.

and may fall when turned, but soon rises. Knuckling of the fetlocks, monoplegia affecting a limb, an ear, an eye, or the throat is a grave paralytic sign. General paralysis often begins with a knuckling of the hind fetlocks that soon leads to loss of support, collapse, and inability to rise. Yet the animals may eat and be thrown into convulsions when handled. Cows may at first show dullness, anorexia, refusal to move, congested mucosae, and finally epileptiform attacks that follow one another until the end. In others the initial symptoms are a partial paralysis of the hind limbs, clonic spasms of the lateral neck muscles, or a retraction of the head and neck. Frequent general symptoms are complete anorexia, congested mucosae, rapid labored breathing, groaning and fast pulse. Loss of condition is rapid. No effective *treatment* has been found, though symptoms of an apparent meningitis may soon

disappear. The course is from two to ten days depending on the extent and degree of the lesions.

ENCEPHALITIS

(Acute Encephalitis; Nonsuppurative Encephalitis; Degeneration of the Brain)

Acute encephalitis is characterized by motor irritation and paralytic symptoms, and by disturbed consciousness, but when the lesions are

Fig. 44.—Encephalitis of the right cerebral hemisphere in a horse. Extensive degeneration with liquefaction and hemorrhage. The chief symptoms were paralysis, especially of the left side, depressed consciousness, and motor irritation (nystagmus).

confined to the cerebellum the consciousness is normal. The anatomical features are degeneration and liquefaction of the brain substance; the disease is infrequent.

Etiology.—This form is commonly attributed to infections or intoxications, but in the few cases observed by the author there has usually been no evidence of the cause. In the horse it may develop in influenza or contagious pneumonia. Hemorrhage and marked congestion of both the meninges and the encephalon have been found in cows as a secondary condition in gastroenteritis.

Morbid Anatomy.—When the lesion is *circumscribed*, the external

surface of the brain may appear to be normal, or there may be an area where the convolutions are flattened and the color yellowish. On section, one or more yellowish foci with indistinct convolutions may be found. The size varies from that of a pea to that of a lobe of the brain. The center may be soft or liquefied. Histological examination shows a small-celled infiltration, probably with lymphocytes, and degeneration of the nerve cells.

In a *diffuse* type, extensive degeneration may involve the hemisphere to such an extent that a cream-like, yellowish white fluid flecked with blood oozes from the surface when the skull is removed. On section, the cut surface is found to be distinctly yellow, and there may be numerous cavities filled with thick fluid debris. The walls of the cavities are congested or hemorrhagic, and numerous spots and areas are distributed over the cut surface.

Symptoms.—The disease may be ushered in with dullness, but usually one first observes signs of an irritative or paralytic lesion, as twitching of the neck muscles, or paresis. In *circumscribed lesions in the cerebellum* there may be tetanic spasms of the muscles of the neck (orthotonus) and limbs, and paralysis, while the consciousness remains normal. If the area is small the animal may show marked improvement for a time, but the changes are progressive and finally lead to complete paralysis and death. More often the lesion is in the *cerebrum*, when one observes a combination of disturbed consciousness, motor irritation, and paralysis. When the paralysis chiefly affects one side of the body, the changes in the brain are on the opposite side; in one case, the left side was paralyzed from extensive degeneration in the right hemisphere. At times there may be a rolling of the eyeball—nystagmus. General symptoms are usually absent; such animals may eat and drink normally, respond to calls at times, and carry a normal temperature and pulse. On attempting to walk, the victim staggers and falls or leans against a wall for support. It remains standing in one position for hours or days, but finally falls and is unable to rise. When the damaged area is extensive, or develops rapidly, death results in a few days, otherwise it may occur only after weeks or months.

In *hemorrhagic meningo-encephalitis* secondary to infections and intoxications, as observed in cows, the dominant nervous signs are paralysis and motor irritation. One finds salivation, the head drawn to the right or upward, one ear drooped, ptosis affecting one or both eyelids, blindness, loss of eye reflexes, the feet drawn under the body, pressing forward in the stanchion, and a tendency to fall when disturbed; finally the animal is unable to stand, though it may still eat and swallow. Death usually occurs within a week.

The *differential diagnosis* of meningitis and encephalitis is assisted by consideration of the following three points: (1) Meningitis is by far the more common of the two, especially in cattle and sheep, where usually it is secondary to some toxic or infectious condition. (2) Meningitis is characterized by various forms of motor irritation. Paresis or paralysis may be marked, but this tends to be general in character and not associated with disturbed consciousness. (3) Encephalitis is characterized by deranged consciousness, and often by a one-sided paralysis (hemiplegia).

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(Toxic Encephalitis in Horses; Cornstalk Disease; Moldy Corn Poisoning; Nonvirus Encephalitis; Leucoencephalitis)

In 1901 Buckley and MacCallum[1] described a brain disease in horses in Maryland to which they gave the name acute hemorrhagic encephalitis, and one year later Butler[2] reported the experimental production with moldy corn of what was apparently the same disease under the term leucoencephalitis. The lesions were unlike those of virus encephalitis; fewer animals were attacked, and the area of distribution was comparatively small. The described symptoms were coma and delirium, muscular twitching, walking in circles, staggering, and paralysis of the throat. Death usually occurred in from 48 to 72 hours. Both authors described extensive areas of softening in the white substance of the cerebrum; these were macroscopic and marked by numerous hemorrhages. It was attributed to eating moldy fodder or corn.

Within the past few years there have been reports of this or similar forms of encephalitis in equines in the Middle West, especially in Illinois, Iowa, and Kansas. Graham[3] has reported that approximately 5,000 horses succumbed to this disease in Illinois in the winter of 1934-35. The disease appeared in October and reached its peak in December. Thus it has a seasonal occurrence distinctly different from that of virus encephalitis. Experimental horses contracted the malady and died in from 23 to 26 days after being turned into a cornfield where natural cases had developed and typical gross lesions were found in the cerebrum. The affection was usually afebrile. The nervous symptoms were dullness, somnolence, sometimes excitement, followed by local or general paresis, unilateral loss of sensation or sight, slow breathing, walking in circles, staggering, and tremors. Jaundice was common. As a rule it was rapidly fatal. This disease is relatively more frequent in a

wet season when corn matures late, early frosts occur, and extensive mold growth results. In general the lesions and symptoms are similar to those shown by the horse whose brain is illustrated in Figure 44. Clinically the disease is indistinguishable from virus encephalitis, but the seasonal occurrence and the general prevalence are different. Efforts to incriminate microorganisms or molds have failed. The microscopic changes in softened areas of the cerebrum have been described by Schwarte and associates.[4] These consist of degeneration, liquefaction necrosis, edema, and hemorrhage in the white substance of the brain. The disease was also reproduced experimentally by Biester et al.[5] by feeding moldy corn, but no clue as to the pathogen involved was observed. According to McNutt[6] the disease is caused by a toxin which causes degenerative changes in the brain but no inflammation, "hence it is not an encephalitis." A similar or identical disease has frequently been observed in horses in the Finger Lakes Region of New York State, "Penn Yan Disease," where it is endemic on numerous farms at all seasons of the year.

In the differential diagnosis between toxic and virus encephalitis, Swan[7] has cautioned against the diagnosis of the virus form until the virus has been identified by laboratory examination. He mentions a number of cases in which the symptoms were like those of virus encephalitis, but the horses recovered following the administration of aloes or pilocarpine; all were on a diet of alfalfa hay. While such cases are not frequent, they present a difficult problem in diagnosis, especially when only one or two animals are affected and recover. In the usual form there are somnolence, paresis, jaundice, suppressed peristalsis, and a medium degree of fever. The gait is slow and unsteady when the horse is made to walk, and backing is difficult. The ears droop, the eyes remain closed and there may be photophobia with lachrymation. In the midst of outbreaks of toxic or virus encephalitis, as well as in cirrhosis of the liver, similar symptoms with recovery occur. Whether this nonfatal condition is an affection of the brain, or a combination of digestive and liver derangement, "toxic indigestion," has not been determined; probably it is caused by some unknown toxic substance in the food, perhaps in alfalfa.

Reports upon encephalitis in the United States and other countries indicate that in respect to etiology and pathology there are various forms of this disease. The symptomatology, however, seems to be very similar or identical regardless of the cause. In addition to viruses that have a special affinity for nerve tissue, the nervous system is exposed to attacks from many agencies that cause deranged functions in other organs. Thus secondary nervous symptoms in domestic animals are

relatively frequent, and it is important to become familiar with the manifestations that indicate deranged cerebral functions as distinguished from symptoms originating in other parts of the nervous system and other parts of the body.

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(Cornstalk Poisoning in Cattle)

Cornstalk poisoning in cattle in Iowa was described by Schwarte et al.[1] in 1939. It is caused by an unknown toxic substance obtained from uncut cornstalks following a drought which prevents normal development of the corn. Cornstalk poisoning in horses, on the other hand, follows an opposite seasonal condition, such as a wet season and early frosts with moldy corn, which is not toxic to cattle. Two cases of cornstalk poisoning were observed in Ithaca after the extreme drought in 1939. On this farm the stalks were cut and run through a shredder in the barn; when the stalks were fed without shredding the disease appeared. Efforts to identify the essential cause as an infection, prussic acid poisoning, or nitrate poisoning have given negative results. In Iowa the feeding of cut cornstalks from affected areas has not caused poisoning; apparently the toxic substance in the drought-injured cornstalks is not stable.

Morbid Anatomy.—Petechial hemorrhages are found in the subcutis, the serous membranes, the epicardium and endocardium, the bladder, the thymus gland and sometimes in the cortex of the kidney. The liver and kidneys are swollen and the latter are degenerated. The spleen is not swollen and lesions have not been observed in either the lungs or the brain. Elongated hemorrhages in the mucosa of the rectum and small colon have been reported.

Symptoms.—In one case observed at the New York State Veterinary College the onset was sudden in the form of progressive dullness with recumbency, staring expression, and extension of the head. An

outstanding symptom was a simultaneous twitching of both ears and contractions of the muscles of the thorax and abdomen. The animal rose with difficulty and when pushed from behind showed a labored staggering gait. The conjunctiva was injected, the iris was completely contracted, and the pupil was visible only as a mere slit; blindness was evident. The pulse, respiration and temperature were normal. The rumen was atonic and peristalsis was suppressed. At the close of micturition there was a prolonged dribbling of urine suggestive of paralysis of the bladder. The Ross test for acetonuria was negative. As the symptoms progressed there was an increase in tetanic contractions involving the limbs, combined with marked drooling and grinding of the teeth. Although the iris relaxed the animal was apparently blind. In a second cow there were lateral curvature of the spine, paralysis of the throat, and violent convulsive movements when down in the stall. The blood calcium, inorganic phosphorus, and magnesium were normal. The course was about 24 hours ending in death. No improvement followed the administration of sodium thiosulfate with sodium nitrite, or magnesium sulfate. While this disease is apparently an intoxication and not an encephalitis, it is described here because of the predominant nervous symptoms, and our lack of knowledge of the nature of the toxic agent. It is differentiated from typical encephalitis by the short course and the absence of fever; the symptoms resemble those of prussic acid poisoning.

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(Sporadic Bovine Encephalomyelitis; Buss Disease)

A form of encephalitis affecting cattle under 3 years of age. It was first reported in Iowa by McNutt and Waller[1] in 1940, described by Boughton[2] in Texas in 1941, and reported from Minnesota. It is apparently caused by an unidentified agent intermediate between the viruses and bacteria. The significant lesions are a serofibrinous pleuritis and peritonitis, and microscopic perivascular inflammation of the brain and cord. The clinical features are continuous high fever, gradually developing depression and emaciation and finally coma.

Etiology.—This disease is relatively infrequent. In 1940 it had been recognized in five herds in the eastern part of Iowa extending 200 miles diagonally across the State. As a rule it affects only a few animals in a herd and the mode of natural infection is unknown. It occurs sporadically but exceptionally it may persist in the same herd for several years. Calves have sickened in from 12 to 14 days after drinking milk from a

sick cow, but they sicken only rarely when kept with the diseased. It has been observed in aged cattle in one herd. The infective material has been recovered from the brain, spleen, lymph nodes, nasal exudate, urine, milk, and from the exudates in the pleural and peritoneal cavities, but not from the blood stream. Calves are artificially infected by subcutaneous or intracranial injections, and guinea pigs by intraperitoneal inoculation; other species are immune. All calves are susceptible to the first exposure and recovered animals are immune. The causal agent has been cultivated continuously in the yolk sac of developing chick embryos for nearly two years.

Morbid Anatomy.—Gross lesions are found only if animals are autopsied after several days' sickness, but there are no significant gross lesions of the brain, cord, lungs, liver, or kidneys. The significant lesions are in the peritoneal or pleural cavity or both. These are serofibrinous exudates with pleurisy or peritonitis. Adhesions are not present. The body cavities contain straw-colored serum in which are large masses of yellow fibrin and thin networks of fibrin. The microscopic brain lesions are perivascular inflammatory reaction, infiltration, and often thrombosis of the small vessels. Thromboses are significant since they are rare in other forms of encephalitis. The nasal passages are congested and contain catarrhal exudate.

Symptoms.—The incubation period ranges from 5 to 27 days, and the course of the disease from 1 to 3 weeks. From the onset there is a progressive depression with emaciation, a continuous temperature of 104° to 107° F., inappetence, a slight diarrhea and a moderate discharge from the nose and eyes. On one farm where aged cattle were affected the usual encephalomyelitis was not produced in a majority of cases; all of the 9 affected animals first showed tenderness of the feet with swelling just above the hoofs, and 7 recovered. The mortality is from 40 to 70 per cent. No treatment has been suggested.

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(Suppurative Encephalitis; Abscess of the Brain)

Abscess of the brain is found occasionally in colts, where it is mainly of metastatic origin in strangles; it is rare in other animals. Pus-form-

ing organisms are often carried to the brain from distant infections, especially in cows, but usually they localize and multiply in the meninges. Very few cases of verified abscess of the brain have been described. Suppurative meningitis may penetrate to the encephalon, but the chief lesions are on the surface. On autopsy, one may readily mistake extensive liquefaction of the brain tissue for abscess formation. The symptoms of abscess are like those of degenerative encephalitis—disturbances of consciousness, motor irritation, and paralysis.

A fracture of the skull following the suture of the sphenoid and occipital bones in a horse caused diffuse suppurative encephalitis with pockets of pus beneath the meninges. The symptoms were nosebleed, bleeding from the ears, incoordination of movements and finally paralysis.

Abscess of the brain in a heifer, described by Gibbons,[1] was apparently metastatic from recurrent abscesses on the left side of the neck, from which a fistula led to the cervical vertebrae. The head and neck were extended and carried to the left; when the neck was straightened and raised the animal would fall, roll on its back, show rolling of the eyeballs, convulsion, and finally resume a normal recumbent position. When moved there was a tendency to circle to the left. Autopsy revealed abscess of the cerebellum and the left half of the cerebrum.

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MENINGO-ENCEPHALITIS IN RUMINANTS

(Circling Disease; Encephalitis in Sheep; Listerellosis)

Definition.—Listerellosis is a highly fatal specific infectious disease of sheep, goats, cattle, and to a lesser degree of swine, caused by *Listerella (Lysteria) monocytogenes*. The characteristic postmortem lesion is a microscopic perivascular round-celled infiltration in the tissues of the brain stem; there are no macroscopic changes. In sheep, walking in circles and paralysis are the distinctive symptoms.

Etiology.—Circling disease was first described in sheep by Gill[1] in New Zealand in 1931, and in 1932 Doyle[2] described microscopic lesions of the medulla of sheep in Indiana. In 1934 Jones and Little[3] described a microscopic round-celled perivascular infiltration in the midbrain and stem of the brains of 13 cows in New Jersey slaughtered on the second to the sixth day after an attack of nervous symptoms. From these lesions a gram-positive rod was recovered. No gross lesions were observed. It is probable that these cows were affected with *Listerella*. It affected only a few cows in a herd but was recurrent in different years.

In New York it was described in cattle in 1935 by Fincher,[4] and in sheep in 1940 by Olafson.[5] In Illinois it has been described by Graham et al.,[6] in Iowa by Biester and Schwarte,[7] in California by Hoffman,[8] and in Oregon by Muth and Morrill.[9] It is widely prevalent in this country. It is the most frequent form of encephalitis in ruminants in the vicinity of Ithaca where it occurs in the months of December to July on certain farms. In sheep it attacks from 1 to 10 per cent of the flock and is usually fatal within three or four days to a week. While it is most prevalent in adults, it may attack spring lambs only 6 to 8 weeks of age. Heavy feeding of silage has been regarded by some as a predisposing cause, but it has been seen in sheep receiving no silage. Artificial transmission occurs in sheep following intracerebral injection of infective material. This causes an acute exudative encephalitis and meningitis with death in from 24 to 36 hours; the symptoms and lesions are unlike those caused by natural infection. Intracerebral inoculation of rabbits causes death in one or two days.

Listerella is a gram-positive rod one or two microns in length that grows readily on artificial media inoculated with infected brain tissue. The habitat and mode of infection are unknown. It has also been recovered from the liver of aborts and of animals which died of another disease. It is suspected that apparently healthy animals may be carriers.

Morbid Anatomy.—There are no macroscopic lesions. The most constant and marked changes are in the brain stem in the form of perivascular accumulations of round or lymphoid cells, and areas of degeneration containing large mononuclear cells. Gill[1] refers to the areas of degeneration as microscopic purulent foci.

Symptoms.—In the beginning there is dullness with a tendency for the affected animal to keep by itself and to wander. Frequently the head is held to one side, or it may be retracted, or held low. If the head is straightened it returns to its former position when released. In the majority of cases there is forced movement in the form of a circle to the right or left, and always in the same direction, though many affected sheep wander until their heads come in contact with a fence or some other obstruction, when they stand pressing their foreheads against it. Monoplegia in the form of paralysis of the lower lip or drooping of one ear is frequent. There is a marked tendency to somnolence. There may be salivation, nasal catarrh, and edema of the conjunctiva of one or both eyes. In the New Zealand cases the blood showed a marked increase in the polymorphonuclear count. An affected sheep may hold a wisp of hay in the mouth for hours.

In the differential diagnosis, pregnancy disease of ewes may closely resemble circling disease. As described by Olafson,[5] the seasonal incidence

and course are the same; facial and ear paralysis are absent in pregnancy disease; grinding of the teeth is more common in pregnancy disease; the head may be turned to one side but does not fly back when straightened as in encephalitis; acetonuria is present from the beginning in pregnancy disease.

The symptoms in swine, as described by Biester and Schwarte,[7] were trembling in large animals, dragging the hind legs, various degrees of incoordination, and stilted tetanus-like movements of the forelegs. The highest mortality occurred in suckling pigs, but some animals that manifested severe clinical symptoms recovered.

Fig. 45.—Meningo-encephalitis in sheep. Note the circling, the paralysis of the lower lip, and the head turned to one side. (Photographs by courtesy of Peter Olafson.)

In the encephalitis in calves, reported by Mathews,[10] 5 animals were affected in a group of 90 in a feed-lot in January. They refused food and water, wandering aimlessly, and at irregular intervals would stand with their heads lowered and pushed against the fence or the side of the barn. Paralysis of the pharynx and coma soon developed. Death occurred after a course of from five to eight days. In the medulla there were distinct perivascular round-cell infiltrations, and areas of degeneration filled with large mononuclear leucocytes.

In the winter and spring of 1934 and 1935 several cases of "circling disease" in cows were encountered in our ambulatory clinic. It is probable that these are identical with cases which have been diagnosed in the past as encephalitis. Reports from veterinarians indicate that the disease is not infrequent in cows in New York State. Typical cases have been described by Fincher.[4] The first was an 8-year-old cow seen February 28, 1934. When released from the stanchion in the morning she

rushed out of the barn in a peculiar manner. When driven back into the barn she walked with difficulty. When examined at night she was pushing forward in the stanchion; this was done so vigorously that at the slightest disturbance her fore feet would slip back and she would fall on her knees. The expression was anxious and wild. There was a complete loss of appetite and milk secretion. The temperature was 104° F. Other symptoms were lacking. The Ross test of the urine for acetone was negative. On the following day there was not much change. The head was carried slightly to the right. The eyes were rolled outward and backward in a fixed position. The pulse was fast and barely perceptible, the respirations 48, and the temperature 102.8. Two days later paralysis developed and death occurred twenty-four hours thereafter. Autopsy negative. The brain was removed and studied by Dr. Olafson.[5] He obtained a gram-positive rod in pure culture from the brain stem. Microscopic examination of sections of the brain stem showed perivascular accumulations of round or lymphoid cells.

The second case was that of a 4-year-old cow. On the night of May 29, 1934, she gave no milk, although fresh for about six months and on good pasture. On the following day she persistently turned the head and neck to the right when in the stanchion and showed a slight tendency to press forward. When released it was necessary to push the cow out of the stanchion, but when she reached the yard she moved rapidly in circles to the right. On reaching a fence, she tried constantly to turn to the right over the fence. At times she appeared to be blind. When forced into the barn she plunged recklessly through a heavy closed door. The eyes were dull, the temperature 104° F., the pulse 72 and the respirations 15. Saliva drooled from the mouth. The digestive tract was normal, and the urine was negative to the acetone test. On post-mortem examination, histologic changes similar to those in the previous case were found. Contaminated media prevented bacteriologic conclusion. Two other similar cases in one herd were seen in 1935. In each instance the temperature was normal, and the brain stems yielded a pure culture of the gram-positive rod.

A first-calf heifer that had freshened two weeks previously showed diminished appetite and milk flow, dullness and constipation, ushered in with staggering and shivering. On examination February 21, excitement, wild expression, and a temperature of 106° F. were found. The urine was negative to the test for acetone, but on the following day it was distinctly positive and a diagnosis of acetonemia was made. On the 23rd the heifer was down and unable to rise; the head was extended, the ears drooping, and the conjunctiva congested. Temperature 100.4° F., pulse 88. Because of disturbed consciousness and paralysis menin-

goencephalitis was suspected. On the 24th there was a slight drooping of one ear, inability to rise and a pulse of 100. On the 27th she was destroyed. Postmortem examination was negative, but *Listerella* was found in the brain cultures. Hyperesthesia has been observed.

A 4-year-old Jersey was found down the evening of March 16 with marked extension of the neck. On the morning of the 17th the cow was recumbent with the head drawn back and attempts to roll the animal on the sternum caused tetanic spasms of the rear limbs. Death occurred within twenty-four hours.

In making a *clinical diagnosis* chief importance attaches to the depressed consciousness (somnolence), motor irritation (muscular twitching, wandering, circling, head pushed against any object), and paralysis, a syndrome which always indicates a disturbance of the brain. While the disease is regarded as invariably fatal, and that has been our experience with cattle, there are reports of recovery following the administration of antibiotics and sulfanilamide. Towards the end of an outbreak the mortality may decline. An attempt by Olson et al.[11] to immunize sheep during an outbreak was a failure.

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CHRONIC DROPSY OF THE VENTRICLES

(Dummies)

This is a chronic disease in which the clinical features are recurrent depression of the consciousness and paresis. There is an increase in the cerebrospinal fluid in the ventricles of the brain. It occurs infrequently in the United States, and appears to be most common among heavy draft horses in the Middle West. A few horses have been treated for "dummies" in our ambulatory clinic, but only one that has come to autopsy has proved to be hydrocephalus. The *cause* is unknown. The *pathological changes* are a marked increase in the size of the ventricles, increase in volume, but not in the weight of the brain, and an atrophy of the brain substance. According to Matthias,[1] primary hydrocephalus is essentially an edema of the brain, in which there is a decrease in the size of the subarachnoid space of at least 40 per cent.

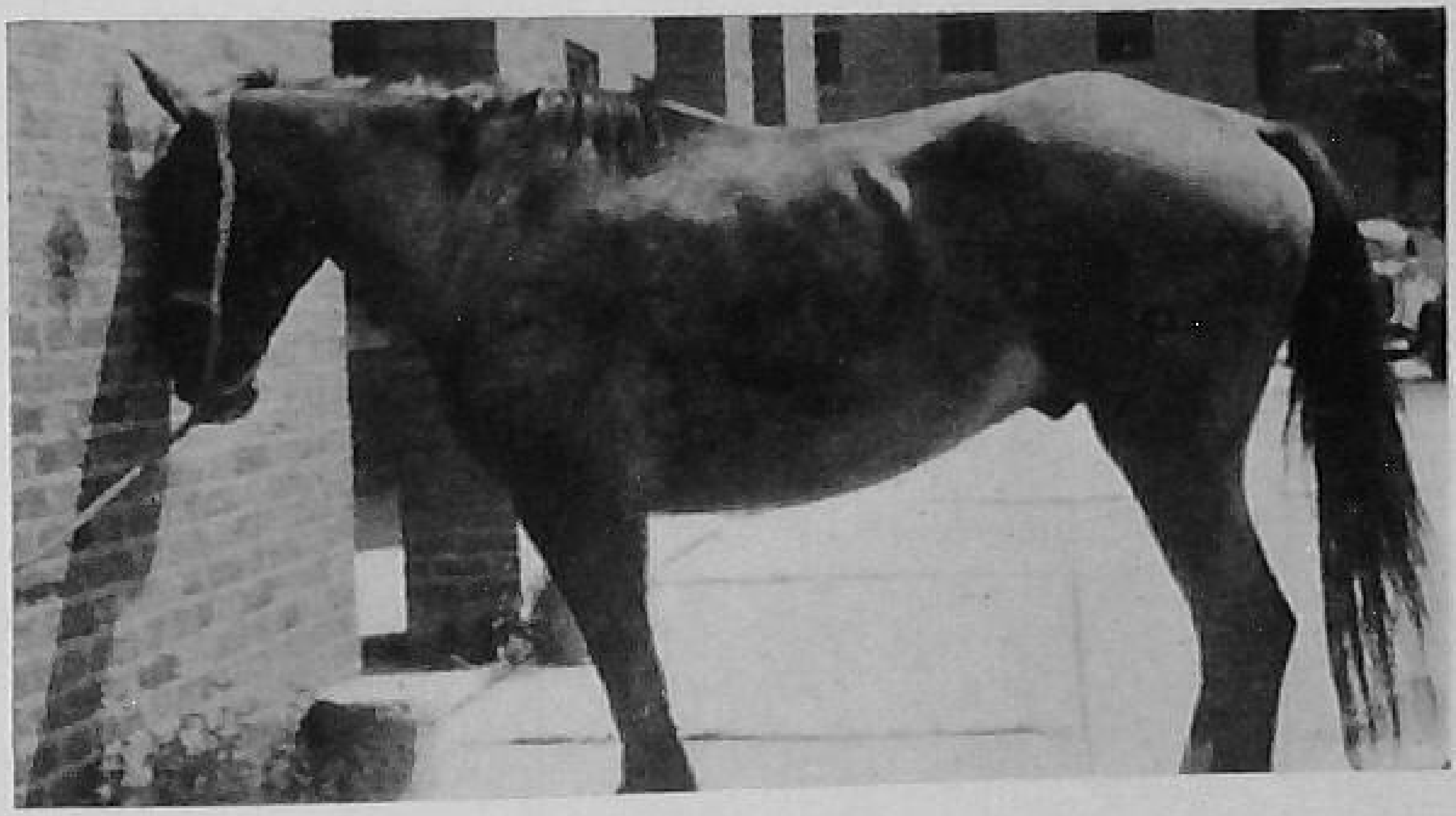

Fig. 46.—Hydrocephalus. (Photograph by W. J. Gibbons.)

Symptoms.—The onset is gradual. Failure to respond normally to the reins is one of the first signs of approaching "dummies." Depressed consciousness is shown by an unphysiological sleepy attitude, with the eyes half-closed and the head lowered when standing; by the lowering of the head deeply into the water when starting to drink; and by standing with half-chewed morsels of hay in the mouth, as if somnolence had interrupted eating. At periods when the symptoms are most marked, there is unsteadiness in walking and the victim may easily fall as if from vertigo. If forced to back, the hind feet are not lifted and backing may be impossible. There may also be refusal to move forward or in any direction indicated by the driver. In addition to these derangements of motility and consciousness, various forms of motor irritation may be present, as walking in circles, pressing the

head against the wall, and lifting the feet abnormally high when walking, as if wading a stream. In advanced cases the sensitiveness may be reduced to such a degree that pressure around the coronet or tickling the inside of the ears brings no reaction. There is also a depression of the digestive functions. The condition is aggravated by work and exposure to heat. If the patient is allowed to remain quietly in a cool place improvement follows. Often, the course finally leads to complete uselessness and the animal is killed; otherwise it may continue over months or years.

In the *differential diagnosis*, one considers other affections of the brain that may, by means of increased intracranial pressure, or otherwise, produce the same symptoms. These are tumors of the brain, especially cholesteatomas, and encephalitis with degeneration, either of which is more frequent in the writer's experience than hydrocephalus. A positive anatomical diagnosis can be made only by autopsy.

Treatment.—Rest, and laxatives of large doses of sodium sulfate (¼ to 1 lb., 250 to 500 Gm.) daily may be followed by marked improvement and in some instances by apparently complete recovery.

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BRAIN TUMOR

Tumors of the brain are infrequent in animals. The type most often met with in our clinic is *cholesteatoma* in the horse. This is a chronic fatty growth from the choroid plexus located in the lateral ventricle. The *symptoms* are much like those of "dummies," or encephalitis. There may be a history of recurrent dummy-like symptoms over a period of six to twelve months. Finally, as the tumor enlarges, distinctive signs of disease of the encephalon appear in the form of a marked depression of the consciousness combined with paralysis. In the case described by Fincher, there was a right-side paralysis caused by a tumor about the size of a goose egg in the left ventricle. *Autopsy* reveals the tumor in the ventricle. There may be a marked enlargement of the lateral ventricle and atrophy of the brain substance, as in hydrocephalus, and a deep yellowish discoloration of the brain tissue, as in encephalitis. When the tumor is large the hemisphere in which it is located may be slightly larger than the other. In one case there was a marked congestion of the entire surface of the brain and hemorrhage in the cerebral cortex; the horse had been affected for months with circling and other symptoms of "dummies."

Fig. 47.—Cholesteatoma of the brain in a horse, Fincher's case.

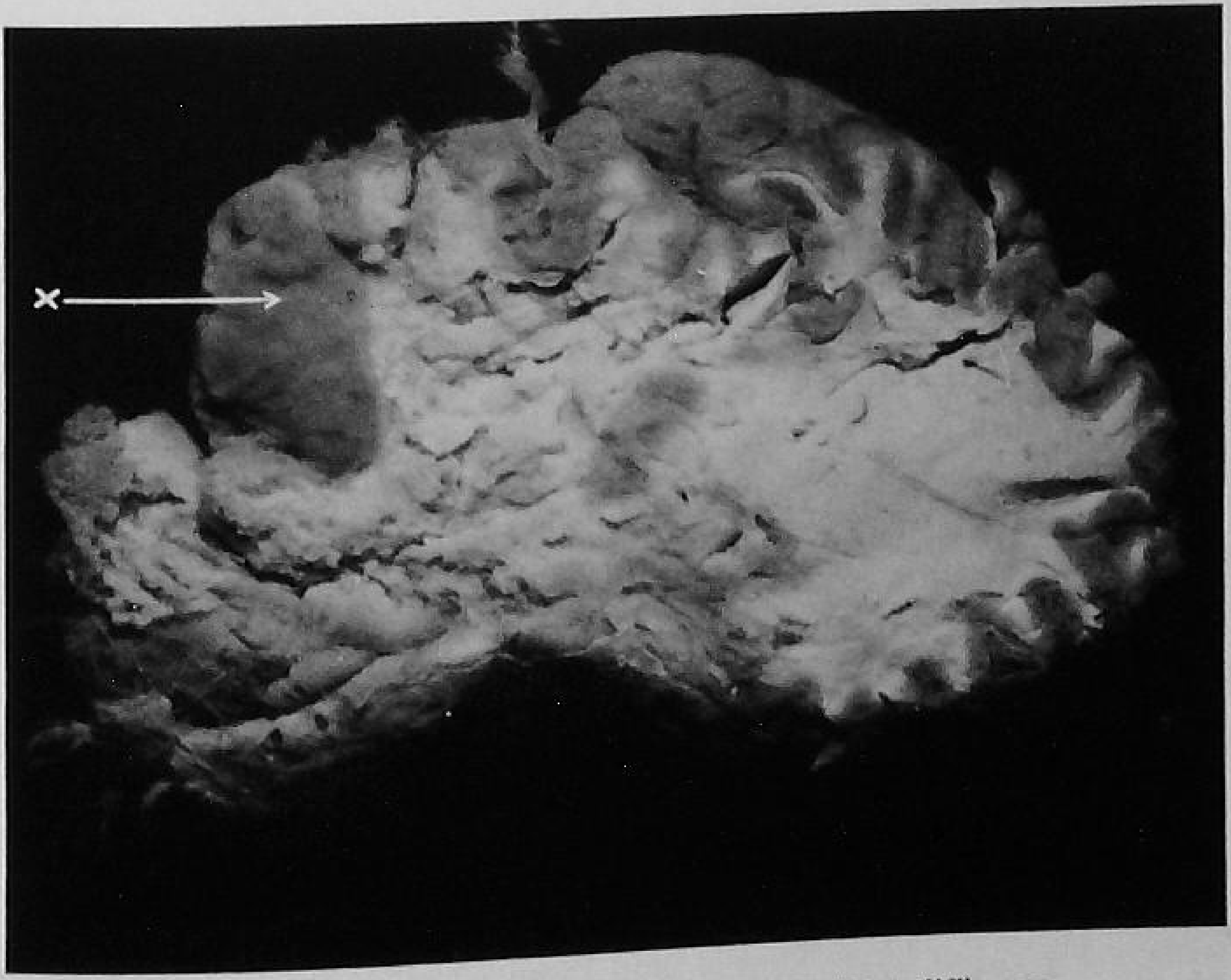

Fig. 48.—Sarcoma of the brain in a cow (X), McAuliff's case.

Malignant tumors may invade the brain by metastasis. In McAuliff's case a large sarcoma appeared at the side of the vulva, and there was a sarcoma about the size of an olive on the extreme antero-lateral part of the left hemisphere. The cow developed a marked paralysis of the right side.

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GID; COENUROSIS

(Staggers; Turnsick)

Definition.—Gid is a chronic brain disease of sheep, less frequently of cattle, characterized by forced movements—circling, rolling—caused by the larval form of *Multiceps multiceps (Coenurus cerebralis,* bladderworm) which develops in the brain, rarely in the spinal cord. The adult parasite is a tapeworm, *Multiceps multiceps (Taenia coenurus)* in the dog.

This disease is met with in Europe, Africa, South America, and Australia, and it is generally distributed throughout the world. It is of frequent occurrence in Western Ireland. In the United States, it was not reported until about 1900, and it has been found chiefly in Montana. In 1900 Ransom[1] reported that "for some reason the gid parasite has never gained a foothold in North America, and until recently, so far as it has been possible to determine, gid has been entirely unknown in this country." In 1909 Boynton and Taylor[2] reported an isolated outbreak in New York State. In 1910 Hall[3] wrote, "It seems certain that the gid parasite was observed in this country as early as 1901." The disease was first reported in Canada by Cameron.[4]

Life History.—In the brain of an infected sheep, the larval form of *Multiceps multiceps* carries tapeworm heads. This larval parasite, the bladderworm, is spherical, from 2 to 4 inches in diameter, and presents a translucent surface having the appearance of a fish bladder full of water. On the surface are white spots, each representing an inverted larval tapeworm head. When the brain of a sheep is eaten by a dog, these heads pass to the intestine where they develop in about a month to the mature tapeworm, 2 or 3 feet long. The posterior segments of the worm contain many eggs; these are round, yellowish or brownish, and 31 to 36 microns in diameter. The posterior segments separate and pass out with the feces and the eggs infect the water or grass. On being swallowed by cattle or sheep, the shells are dissolved, the small embryos enter the blood vessels, and those which lodge in the brain or

spinal cord develop in from seven to nine months into the mature bladderworm. Tapeworm eggs are not highly resistant, and sheep are infected only with those that have been recently dropped. Wet weather favors infection; the eggs soon perish when exposed to dryness and heat.

Symptoms.—The disease has two stages. The first is an acute meningo-encephalitis caused by migration of the embryos when they first reach the brain shortly after the ingestion of the eggs. As a rule this stage does not occur, or is not recognized, and is rarely fatal. The symptoms are fever and restlessness at pasture ten to fourteen days after infection. Mild cases show a depressed consciousness; the affected animals remain behind and dislike to move, they rarely circle. In more severe forms there are marked depression, pain on pressure over the skull, wild expression, restlessness, and sometimes strabismus. There may be tonic spasms of the neck muscles, and even convulsions.

The second and final stage usually appears in the winter months after a wet season favorable to the survival of the eggs. The dominant symptoms fall under the classification of motor irritation; apparently they are caused by an increase in the intracranial pressure. The affected animal pivots around the hind legs, walks in circles, or stands with the nose between the forelegs; the nose may be carried back so far that the sheep keels over. At other times there may be rolling movements; thus the body may rotate around either the longitudinal or transverse axis. Standing with the head pressed against objects, or depressed and to one side, are frequent positions. Periods of excitement and depression are frequent; these are manifested by wandering or running in an aimless manner or standing for long periods in one position. With the development of the bladderworm, pressure on the skull may cause softening and bulging of the bone. These symptoms are a guide for the location and time of operation in countries where gid is of frequent occurrence in sheep and cattle. Unless relieved by means of an operation, the second stage of the disease usually terminates in death in from one to two months.

Prophylaxis and Treatment.—In localities where the disease is not frequent, as in North America, the only logical method of control is to destroy the brains of slaughtered or dead sheep in order that they may not be consumed by dogs, coyotes, and similar predatory animals. All sheep that are known to have the disease should be slaughtered and their brains destroyed. An additional prophylactic measure is to administer a taeniafuge to suspected dogs. Operative removal of the cysts is said to be successful in about 50 per cent of the cases. The skull is trephined, the dura mater incised, and the bladder punctured. According to O'Brien,[5] all ruminants in Western Ireland showing progres-

sive chronic brain disturbance are presumed to be affected with either tuberculosis or gid. O'Brien[5] describes the operation with the use of a special boring trocar, and states that the vast majority become perfectly normal.

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BULBAR PARALYSIS

(Glosso-Labio-Laryngeal Paralysis)

The term bulbar paralysis has been applied to various diseases characterized by paralysis of the pharynx and other organs supplied by the cranial nerves, vi-xii, that branch from the medulla oblongata. This form of paralysis is symptomatic of encephalitis, encephalomyelitis, and of intoxication affecting the central nervous system; it is also symptomatic of certain diseases of the peripheral nerves, as botulism. Paralysis of the pharynx also occurs in the horse as an apparently independent malady, the nature and cause of which are unknown. In man there is a bulbar paralysis resulting from an independent chronic disease of the medulla, but according to Osler it has practically no independent existence, since the spinal cord is sooner or later involved. It is doubtful that this condition or any other independent form of bulbar paralysis exists in animals. It is probable that the majority of diseases described under this title are either encephalitis or belong in the group of paralytic cases with undetermined cause and pathology.

SPINAL MENINGITIS

This is a rare disease in domestic animals. In the personal experience of the author, only two cases have been recognized. One was an attack of acute glanders in the cervical region in a horse. The animal had been losing in condition for several weeks. When examined, he showed marked stiffness of the body and limbs, and on his being turned the spine remained rigid. When the finger was placed over the pulse at the jaw, the animal reared violently from pain, and a slight touch over any part of this region was painful. The second case was caused by

a deep strangles abscess above the lumbar vertebrae. Convalescence seemed to be nearly complete when a gradual stiffness and a loss of condition developed, until finally the horse was unable to rise. Prouse and Fitch[1] have described a case of chronic productive pachymeningitis in the region of the seventh cervical vertebra in a horse. The animal was at first lame in the left front leg and apparently recovered; then the right leg became lame. At the end of three months the appearance of the head and neck resembled that of tetanus. Pain and stiffness were progressive until the end of about five months when the horse was killed.

Symptoms.—The most constant symptom of this disease is stiffness, marked by a peculiar rigidity of the muscles. There is also pain and in some instances hyperesthesia. The presence of the latter, induced by slight stroking of the hair or slight pressure, is regarded as pathognomonic of spinal meningitis. Hyperesthesia on stroking the hair is sometimes observed in milk fever in cows, when this disease is complicated with acetonemia. Muscular twitching and general muscular soreness are mentioned by Sonnenberg,[2] who described an enzootic of nonfatal cases associated with laminitis in the horse. Finally, paralysis may develop, when the animal is no longer able to rise. In compression of the cord, paresis occurs early and other symptoms of meningitis are absent. In acute infections of the meninges, the course is about a week, terminating in death; when the lesion is local and of slow development, the course is several weeks. There is no treatment.

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SPINAL MYELITIS

Spinal myelitis is a rare disease, but it seems to be somewhat more frequent than spinal meningitis. In affections of the cord, there is more or less involvement of the meninges, and in enzootics of myelitis certain individuals may also show symptoms of encephalitis.

Etiology.—It may be secondary to the acute infections, as pneumonia, strangles, and influenza in the horse. Infection appears to be the chief cause. In the enzootics in the horse described by Schlegel,[1] it was attributed to a streptococcus, while Fröhner[2] was able to transmit a similar affection by means of blood inoculation. In 1917 Dr. E. M. Pickens and the author observed an enzootic in cows; in a 40-cow dairy

there were 15 deaths in one stable during the winter; the cause was not determined.

A symptom of degeneration of the spinal cord is common in thoroughbred foals and yearlings in Kentucky and Virginia and it is also said to occur frequently in the South and Southwest. Common names for this condition are "wobblers, jakeleg, and kinkback." The predominant symptom is a gradually developing paresis of the hind legs, and in about 50 per cent of the cases observed by Errington[3] there has been a similar lack of coordination in front. The condition is chronic and incurable. Affected animals are worthless. The cause is unknown. It has been suggested by Dimock[4] that the disease is genetic in origin.

Morbid Anatomy.—Macroscopic changes are often absent, though there may be a turbid or reddish spinal fluid. In acute cases, the affected segment may be colored red from hyperemia of the meninges. Areas of degeneration may be found on histological examination of the cord, but there has been little work of this kind in veterinary medicine. In the reported cases the diagnosis has been based on the clinical symptoms or the gross lesions.

Symptoms.—In the enzootic observed by Pickens and the author the most uniform initial symptom was paresthesia manifested by licking the coronary region of the hind feet. At about the same time, one or both hind legs knuckled slightly at the fetlock, and one front leg might be held in an abducted position when supporting the body. Slight curvature and elevation of the tail was a constant early symptom. Twitching and trembling of the muscles in the region of the stifle, and restless movements of the hind feet were frequent. Finally, both sensory and motor paralysis developed in the hind limbs and extended more or less rapidly forward, leading to death in from three to fourteen days. The appetite, digestive functions, and milk secretion remained normal even after there was distinct knuckling of the fetlocks and curvature of the tail. In one or two of the cows of this group, the disease was ushered in with excitement verging on delirium, which led to suspected rabies. *Autopsy* revealed marked inflammation of the cord in the lumbar region. Paralysis of the rectum and bladder was present in the horses described by Fröhner, but the mortality was low.

Treatment is without effect in well-marked acute attacks. The animals described by Fröhner were supported in slings, and the feces and urine were removed manually until the paralysis finally receded and convalescence was complete.

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2. Fröhner, E., Infektiöse Ruckenmarks- und Gehirnlahmung bei Pferden, Berl. tier. Wchnschr., 1924, p. 215.
3. Errington, B. J., The condition called "wobblers," Vet. Bull., U. S. Army, 1938, **32**, 153.
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COMPRESSION OF THE SPINAL CORD

Etiology.—This condition is met with occasionally in large animals, where it is the most common pathological condition affecting the cord. Among the causes, *abscess* formation holds first place, especially in calves and lambs, where it is sometimes found in the lumbar region in

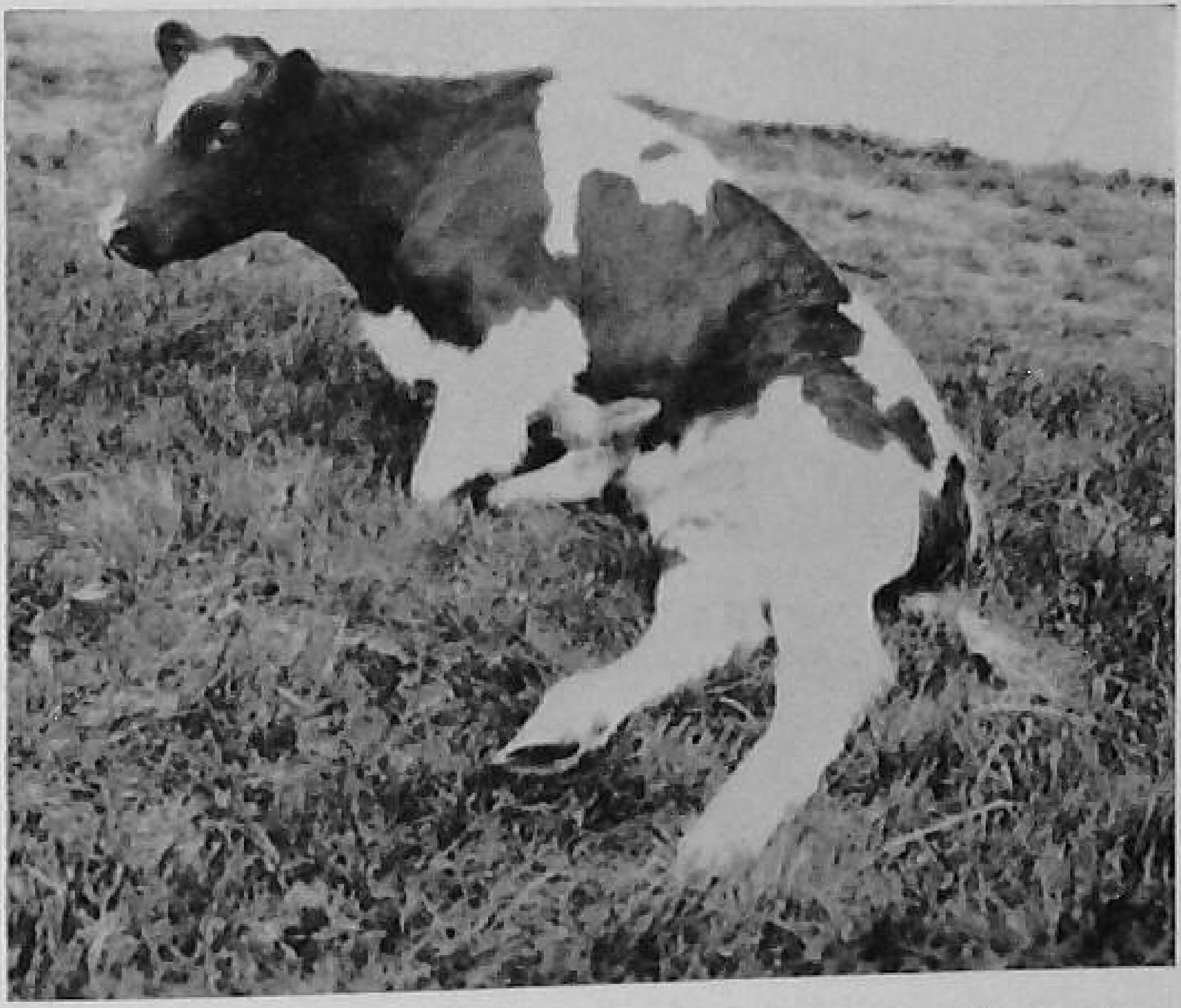

Fig. 49.—Compression of the spinal cord from an abscess in the sublumbar region (navel-ill).

navel-ill. In one instance, a strangles abscess in the lumbar region involved the cord; in a second, a metastatic abscess developed at the eighth thoracic intervertebral space in association with metritis in a cow;[1] in a third, a large tuberculous abscess compressed the cord at the atlanto-occipital joint. Kaay[2] has described a spinal abscess in a cow that was metastatic from a suppurative kneeboil. In progressive sensory and motor paralysis of the hind legs of an 11-year-old bull, Karlson and Boyd[3] found extensive fibrosis covering the fused bodies of the last six thoracic vertebrae; on the ventral surface there were numerous fistulous tracts from which pure cultures of *Corynebacterium pyogenes* were recovered. In a 5-year-old cow having for 9 months shown rigidness of

the neck combined with difficulty in taking fodder and progressive atrophy of the musculature of the thoracic limbs, an enlongated glioma was found in the cervical region—Christensen,[4] Denmark. I have recently observed similar symptoms in a horse which were caused by a purulent arthritis between the last cervical and the first thoracic vertebrae; this animal lowered the head with difficulty, and there was a distinct restriction of the forward movement of the front limbs when walking. A *melanotic tumor* caused paralysis in one of our patients, a 16-year-old grey mare; it penetrated the spinal canal from the third to

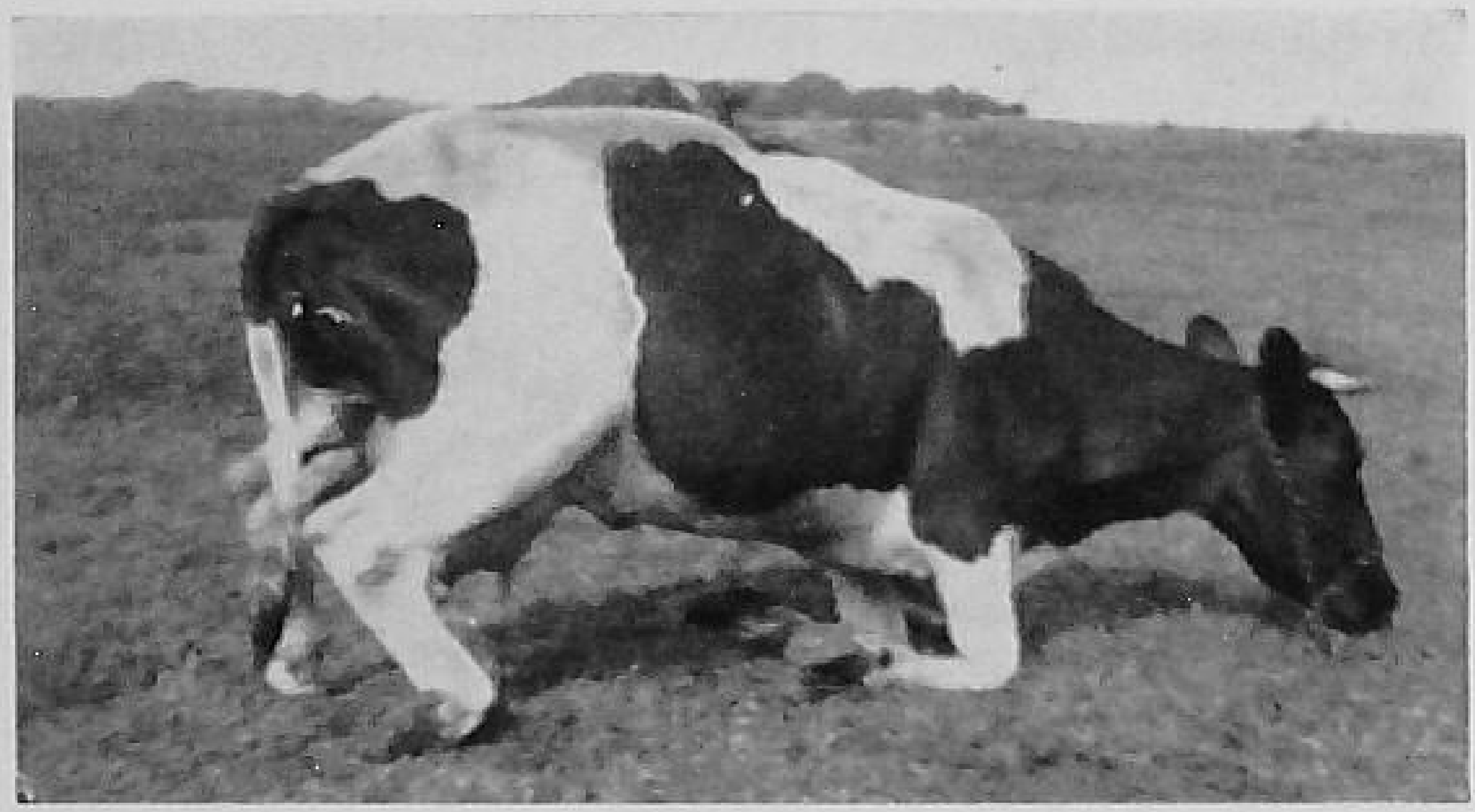

Fig. 50.—Compression of the spinal cord from tuberculosis of the spine. (Photograph by courtesy of Dr. Gustav Danelius, Sweden.)

the fifth thoracic vertebrae. *Tuberculosis* of the vertebra is a relatively frequent cause in swine. Marshall[5] has described a case of spinal compression caused by what appeared to be a hydatid *cyst* at the level of the fourteenth rib in a horse. Hull and Taylor[6] have reported on abscesses affecting the central nervous system of sheep, several of which caused compression of the cord. Compression of the cord from fracture of the vertebrae is not infrequent in *rickets* in swine and it has been observed in experimental rickets in young cattle. In rare instances *bony growths* from the vertebrae press upon the cord.

The *symptoms* consist in a progressive paralysis over weeks or months except in navel-ill or fracture when the onset is sudden. Pressure in the thoracic or lumbar region first causes weakness in the hind quarters—paraplegia. After a time the victim can rise with difficulty or only with aid, and it may assume a dog-sitting position with the hind legs extended laterally in a helpless position. Fecal and urinary evacuations may be suspended. Finally, recumbency becomes permanent and the animal is destroyed.

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DISEASES OF THE PERIPHERAL NERVES

With the exception of the pharyngeal and the vagus, diseases of the peripheral nerves in animals are chiefly of surgical importance.

PARALYSIS OF THE GLOSSOPHARYNGEAL NERVE

Paralysis of the glossopharyngeal nerve is rare as an independent disease. Acute paralysis of the pharynx is usually a symptom of inflammation of the central nervous system, as encephalitis; or it may be due to paralysis of unknown cause or botulism or inflammatory changes or injuries affecting the pharynx directly, all of which are discussed under diseases of the pharynx.

PARALYSIS OF THE VAGUS NERVE

Paralysis of the vagus nerve is met with (*a*) in paralysis of the recurrent nerve, the anatomical basis of "roaring" in horses, where it is generally attributed to degeneration of the nerve as a sequel to the acute infections—strangles, influenza, and pneumonia. (*b*) A *toxic* form results from eating certain varieties of peas, especially *Lathyrus sativus* introduced into Europe from India. This form of laryngeal paralysis may be associated with paralysis of other parts of the body and with deranged consciousness; it is not relieved by operative treatment. (*c*) Lead poisoning in the horse causes degeneration of the vagus nerve; this has been described by Thomassen,[1] Haring and Meyer,[2] and Macindoe.[3] There are two clinical forms:

(1) *Paralysis of the larynx* is met with in animals exposed to fields or streams contaminated from mines or smelters. The distinctive symptom is intense inspiratory dyspnea on exercise. When the animal comes to rest the labored breathing perists much longer than in the usual form of paralysis of the recurrent nerve.

(2) *Aspiration pneumonia* results from the foregoing paralysis when

it becomes extensive enough to favor passage of food particles into the larynx and trachea. It leads to weakness, emaciation, and abscess formation in the lungs. The condition is caused by degeneration of the nerve tissue and when well established is incurable. The course is indefinite when confined to the larynx. After the development of aspiration pneumonia the course tends to be chronic, ending in death after several weeks. The disease is infrequent and is readily prevented.

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FUNCTIONAL DISEASES OF THE NERVOUS SYSTEM

A number of functional diseases of the nervous system, neuroses, in domestic animals have been described. It is probable, however, that in most instances these nervous derangements are secondary to toxic or infectious conditions originating outside the nervous system; the animal recovers and the primary disease is not recognized.

VERTIGO

(Stomach Staggers)

This term is applied to a rare affection observed in driving horses in summer. It is generally attributed to an intoxication or irritation originating in the digestive tract. In some instances it may be either symptomatic of an affection of the brain, or of an intoxication of unknown origin. Apparently it is not due, as in man, to derangement of the auditory nerves.

The *symptoms* consist of attacks of falling when the animal is being driven, or when it first starts on a trip. The horse stops, makes an effort to remain standing, finally plunges down, and lies quietly. Consciousness is lost for three to five minutes, when the victim lifts its head, rises, and seems to be entirely normal. These attacks appear repeatedly and they appear more often when the work is heavy and fast. Between attacks, the condition is entirely normal. *Relief* is usually obtained by restricting the diet, giving light exercise, and administering small doses of salts and bitters.

EPILEPSY

(Blind Staggers)

Repeated convulsive seizures occur in horses, and sometimes in

cattle; these persist throughout life and are not readily prevented. Like vertigo, which it may closely resemble, the disease is rare. Symptomatic convulsions due to toxemia, poisoning, infection, and disease of the central nervous system—meningitis—should not be termed epilepsy. A horse subject to "fits" is a dangerous animal, for a violent attack may appear without warning, endangering the driver, and other animals. As a rule there are premonitory signs. The victim appears restless and tosses the head as if annoyed by a bee. There may be twitching of the ears, eyelids, and muscles of the face and neck. Finally, clonic spasms become general, the animal plunges aimlessly, and goes down in a general convulsion. There may be rapid chewing movements and frothing. The attack lasts not more than five minutes when the animal rises and seems to be normal. The *treatment* is like that for vertigo—light exercise, light diet, and laxatives combined with bitters.

Epileptiform attacks in a group of Brown Swiss cattle were attributed by Atkeson[1] and associates to inheritance. The symptoms were tongue chewing, foaming at the mouth, and collapse; the attacks were relieved by intravenous injections of calcium gluconate.

Reference

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DISEASES OF THE SKIN

PRURITUS

(Itching)

Pruritus is a functional derangement of the normal skin with constant or periodic itching.

Etiology.—It may be secondary to chronic bowel catarrh, irritation from parasites in the rectum, or deranged conduction of the peripheral nerves, as in rabies, pseudorabies, ascending myelitis, and meningitis. In cows, acetonemia may be associated with intense itching around the feet, and similar symptoms may be met with in infections and intoxications. In the diagnosis one needs to be certain of the absence of parasitic and inflammatory diseases of the skin. It may be impossible to determine the underlying cause. Pruritus is the most characteristic early symptom of pseudorabies in cattle, and it is a frequent early symptom in rabies in the horse.

Treatment.—Itching caused by intestinal catarrh is relieved by laxatives and bitters:

℞ Sal Carolina factitii	℥ xvi (500 Gm.)
Pulv. gentianae	
Pulv. nucis vomicae	aa ℥ viii (250 Gm.)

M. Sig.—Tablespoonful (15 Gm.) three times daily.

Local itching is relieved by a 3 per cent alcoholic solution of salicylic acid or camphor. The author has had excellent results with a mixture of sulfur iodide* in oil (1:8) 50 parts, alcohol 40 parts, and formalin 10 parts. Compresses of hot water or alcohol are useful. One part of phenol to 50 of lime water may be effective.

HYPERHIDROSIS

(Sweating)

This is a functional derangement of the sweat glands, having no external cause; it may be local or general.

Etiology.—*General sweating* develops secondary to the accumulation of carbonic acid in the blood in the course of certain circulatory and respiratory diseases; it is also associated with collapse, rapid decline of fever, uremia, cholemia, and convulsions. *Local sweating* occurs secondary to lesions or compressions of the peripheral or sympathetic

* To prepare sulfur iodide: triturate 20 parts sulfur with 80 parts iodine; place in a bottle and set the bottle in a water bath until fusion occurs. After cooling the bottle may be broken and the fused mass pulverized.

nerves, and to inflammation of the spinal cord or its meninges. It has been observed in the vicinity of broken ribs and other fractures. As a rule the underlying cause cannot be recognized. For the control of local sweating, belladonna administered internally is recommended, but it is of doubtful value. A form of "sweating sickness" in calves in Kenya[1] Colony was cured with baker's yeast.

Anhidrosis, inability to sweat, has been described by Gilyard[2] in race horses imported into the tropics. It is a chronic condition associated with sustained loss of blood chlorides under prolonged exposure to excessive heat; it was corrected by adding half a teacup of salt daily to the grain.

References

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HEMATIDROSIS

(Sweating Blood)

In diseases with a tendency to hemorrhage, drops of blood may appear on the skin, thus presenting the appearance of "sweating blood." This is a mixture of blood with sweat due to hemorrhage into the sweat glands; it has been seen in severe attacks of bracken poisoning in cows, in septicemia, and in purpura. Hematidrosis in a horse has been described by Heinrich,[1] who attributed it to *Filaria hemorrhagica*. It is probable that most cases of blood sweating are symptomatic of diseases showing extensive internal hemorrhage. In June, 1916, the author observed one case in which blood oozed freely from various parts of the skin of a pastured cow, which soon died. Extensive internal hemorrhage was found on autopsy. Although an abundance of fresh material was taken to the laboratory, no diagnosis could be made.

Reference

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ALOPECIA

Alopecia is a loss of hair, wool, or feathers with no evident disease of the skin. *Primary alopecia* is rare. *Secondary alopecia* may result from the following causes: (*a*) malnutrition; (*b*) eating spoiled food; (*c*) gastrointestinal catarrh; (*d*) parasitism—flukes, lungworms; (*e*) infectious diseases, such as influenza, pneumonia, strangles, and hog cholera; and (*f*) metallic poisoning, such as mercury or arsenic. A congenital absence of hair in calves and pigs[1] results from iodine insuffi-

ciency. Excessive moisture may cause hairless spots on legs of horses. The habit of wool-eating in sheep, and feather-pulling in poultry, is an occasional cause of bareness of skin in these animals. Shedding of the hair may leave a bare skin when the new hair fails to grow promptly.

In calves 2 to 4 weeks of age almost complete loss of hair within a week has been observed. This was combined with rapid loss in condition, and on one farm was repeated in successive years. The condition started with the presence of a few scaly patches along the back and within 24 hours the calf had rubbed and licked off half its hair, leaving a red sensitive area. The cause was undetermined, and regrowth of the hair took place in about a month. In another instance calves at pasture lost a greater part of their hair from an undetermined cause. *Congenital epithelial defects* with absence of hair on the lower limbs adjoining the hoofs have been described by Hadley et al.[2] Several Ayrshire calves with this defect from a single herd have been observed in the College clinic at Ithaca.

Treatment.—Numerous remedies have been described: frequent washing with soap and water or alcohol; tincture of cantharides (1) to alcohol (5); resorcin (5 per cent); and creolin (5-10 per cent). It is doubtful if any of these add to the benefit obtained by grooming. When the skin has been left bare, thickened, and dry from the effects of mange or eczema, the condition is improved by applications two to three times a week of sulfur iodide in oil (1:8-10), or of warm linseed oil. Iodine deficiency causing goitre and loss of hair in newborn pigs is prevented by feeding pregnant sows potassium iodide at the rate of 5 grains once a week.[3]

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PITYRIASIS

(Dandruff; Seborrhea)

Definition.—An accumulation of bran-like scales on the skin due to excessive secretion by the sebaceous glands; it is caused by various disorders and is a symptom rather than a disease.

Etiology.—It occurs: (*a*) in poorly nourished animals; (*b*) as a secondary condition in eczema and mange; (*c*) in infectious diseases;

(*d*) in chronic digestive disorders; and (*e*) as a symptom of poisoning from iodide of potash.

Symptoms.—Dandruff is a common condition at the base of the mane and tail of the horse, but except for lesions caused by rubbing there is no other symptom. It is also common in long-haired, poorly groomed animals. It is by far the most abundant in animals affected with mange, and when such animals fail to show itching it may be mistaken for seborrhea, especially if the exudate is moist. In eczema and mange, one finds changes in the skin, and in mange itching is prominent.

Treatment.—When the skin is dry, cleanse with soap and water, or gasoline, and apply antiseptics: Acidi salicylici (5), picis liquidae (25), sapo mollis (150). Liquor calcis (50), phenol (1). Acidi salicylici (1), glycerini (3), alcoholis (60).

When the skin is moist—seborrhea, cleanse with gasoline, clip the hair, and apply alcoholic antiseptics. When this form is distributed over the body it is strongly suggestive of sarcoptic mange, even though the parasite may not be readily found.

URTICARIA

(Hives; Nettle Rash)

Urticaria is a transient edematous swelling of the skin of sudden appearance. In cattle the swellings are diffuse, affecting chiefly the region of the eyes, vulva, and teats; in horses they are circumscribed, affecting chiefly the head, neck, and body.

Etiology.—It is frequent in cattle, occasional in equines, and infrequent in swine. In our ambulatory clinic 62 per cent of the urticaria in cattle has occurred in the months from March to May. This seasonal occurrence, and the similarity of the symptoms to those of anaphylaxis caused by the larvae of warble flies,[1] suggest that urticaria in bovines may be an anaphylaxis due to the resorption of toxic material from larvae ("grubs") beneath the skin. In one cow the attack developed immediately after the farmer had squeezed grubs from the skin of the back. Other alleged causes are irritation from cold winds, nettles, blisters, and toxic material from food or infection.

Symptoms.—In *cattle* the onset is sudden and the course is so brief that some of the symptoms have usually disappeared before the arrival of the veterinarian. The owner may report restlessness, fast breathing, trembling, and sweating, but often these are absent. Edematous swellings of the head are common—the muzzle, ears, and with few exceptions the eyelids. Next in frequency are the vulva and anus, as well as

the teats and udder. Swellings may also develop in the submaxillary region, on the throat, on the shoulders and buttocks, along the back, or on any other part of the body. In the beginning, mouth-breathing, salivation, and snoring sounds may lead the owner to suspect choke. The edema may locate in the mucosa of the upper respiratory tract, especially in the nose and throat. The skin of the neck, buttocks, and shoulders may be wrinkled and the hair over these parts erect. Itching may be intense in urticaria of the teats or vulva. The swellings disappear in from six to twelve hours.

In *horses* there is no seasonal occurrence, and in most cases no suggestion as to the cause. Usually the swellings appear suddenly without previous symptoms, but there may have been dullness for one or two days. The elevations are circumscribed, firm, round, about one-fourth inch high, and one-half to one inch in diameter. They may be arranged so thickly that the margins are in contact with one another, and they may become confluent. The usual locations are in the submaxillary region, on the neck, and over the body and hips.

Treatment.—The brief and favorable course renders treatment unnecessary except for distressing symptoms. Itching is relieved by the application of ice water. Atropine sulfate 1/4 to 1/2 grain (0.15 to 0.3 Gm.), or adrenalin 3 to 5 cc. are useful to control dyspnea.

Reference

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ECZEMA

This is a widely variable superficial inflammation of the skin. Usually it is classified for description as erythematous, papular, vesicular, pustular, moist, dry, acute, chronic, parasitic, etc. As it occurs in domestic animals, several of these conditions are present in the same case.

Symptoms.—In the horse, eczema is usually *chronic.* An eruption of papules, vesicles, and dandruff, associated with itching, may develop on any part of the neck and body, at the base of the mane and tail, and less often on the head or limbs. It is most common in the spring, especially in old horses; the first signs are recognized in February and it becomes well-marked in April and May. The eruption may be either local or general; sometimes it spreads extensively from an area of local inflammation, as a blister, a harness pad, or the saddle. The skin is dry, often the hair is long, and usually dandruff is abundant. Itching with consequent rubbing may leave bare spots. Occasionally it attacks several horses in the same stable, as if contagious. Application of ir-

ritants to the skin, such as fly repellants, or coal tar disinfectants, may set up an inflammation that closely resembles mange in extensive loss of hair, presence of scabs and scales, and general appearance. Differentiation is made by the absence of mites and contagion, as well as by the fact that it occurs in the summer months when mange is less active.

Acute eczema in the horse develops rapidly, usually in the spring; in two or three days it may spread over the neck, body, and upper limbs. There are wrinkled areas, papules, scabs, possibly absence of dandruff, and intense itching. It is less frequent than the chronic form. Sudden eruption over a large area differentiates it from mange. An

Fig. 51.—Eczema.

acute form may develop on the limbs and abdomen from neglect of the skin after driving in mud or snow.

Scratches is a special form of inflammation that appears on the flexor surface of the fetlock region, chiefly in horses. Depending on the depth and intensity it may be either an eczema or a dermatitis. Occasionally it extends as high as the carpus or tarsus.

Hair-Root Infection (Sycosis) occurs often in horses at the seat of the collar on the dorsal surface of the neck. The infection may penetrate deeply, causing fistulae. This type is associated with extreme tenderness of the affected part and it may seriously impair the service of the animal for a time. It may be prevented by the use of a well-fitting collar, or a hair pad. If the infection is limited to the hair roots, benefit is obtained from the application of the following antiseptics directly after removal of the collar: 1:1000 alcoholic sublimate, 500 cc.; tannic acid, 30 Gm.; and pyoktanin, 1 Gm. The following powder is also useful when applied previous to adjustment of the collar or

directly after its removal: copper sulfate 3 ounces (90 Gm.), powdered alum 2 ounces (60 Gm.), tannic acid 2 drams (8 Gm.), triturated camphor 2 drams (8 Gm.), and wood charcoal 1 ounce (30 Gm.). Bismuth formic iodide is an excellent dusting powder for this condition.

In *cows* inflammation of the skin caused by irritant disinfectants, such as are sometimes used for lice, causes loss of hair, moisture, and dandruff. A moist type with loss of hair may occur in the folds of the skin between the body and the limbs. There may be loss of hair around the eyes and on the neck, leaving bare patches covered with scabs; this type may prove to be ringworm. Loss of hair with itching and rubbing may be caused by lice. Dermatitis may spread over the abdomen, udder, and teats; it may appear on the back in the form of circumscribed lesions from one-fourth to one-half inch in diameter, or on any part of the body, limbs, or teats, in the form of cracked, bleeding, and scabby areas. It seems to be most common in the cold seasons. Moist acute eczema is more common in cattle than in horses.

An eczematoid dermatosis of the ears, cheeks, neck, and shoulders in young calves indicating intimate connection with ascorbic acid has been described by Cole et al.[1] Improvement followed an initial subcutaneous injection of 3 Gm. ascorbic acid.

Treatment.—Eczema responds promptly to treatment. An excellent remedy is sulphur iodide (1) in light cottonseed oil (Wesson's cooking oil) or olive oil (8-10). When the hair is matted or itching is marked, or the area to be covered is extensive, this mixture is improved by adding alcohol (40 per cent) and formalin (5 per cent). Vienna tar liniment: tar and sulfur (aa 1), green soap and alcohol (aa 2); or lime-sulfur powder (1) in water (40) are useful in the chronic form. Zinc oxide (60) to olive oil (40) is especially useful in scratches.

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DERMATITIS

This term applies to affections in which the deeper layers of the skin are attacked. It frequently develops on the limbs of horses and cattle that habitually wade through filthy mud or manure. In the military camps during World War I deep forms of inflammation prevailed widely among horses and mules more or less constantly exposed to mud, slush, and infected soil. Rope burns are common among horses and mules in camp. Applications of strong disinfectants or of liniments under a bandage may result in severe inflammation of the skin. The

fetlock and coronary regions of cattle may be attacked, in some instances as an extension from an infected pododerm—"fouls." In one instance, large blisters formed on the teats of a cow which also showed dullness and gave a diminished milk flow. In another cow a vesicular eruption appeared on the ears; the muzzle showed superficial sloughing, and the nasal mucosa was covered with scabs. There was also a loss in condition, swelling of the limbs, and eyelids, and marked swelling of the vaginal mucosa. This case is suggestive of dermatitis associated with aphthous stomatitis, which may occur in the absence of mouth lesions. In cows a moist eruption, having the appearance of superficial ulcers, may extend from the udder over the ventral surface of the body. In cows there also may develop a form of dermatitis between the upper part of the udder and the thigh, and because of the odor this may become highly offensive. It responds to treatment with daily application of a solution of 1½ ounces copper sulfate in a quart of vinegar; or, wash daily with 1 per cent creolin solution, dry, and apply Iodex ointment.

Other causes of dermatitis that have been reported are *Staphylococcus aureus* infection in sheep,[1] low ascorbic acid in calves,[2, 3] and nematode infection in cattle.[4] "Lumpy skin disease," believed to be caused by a virus, is a cause of serious economic loss in cattle in South Africa.[5] Skin eruptions occurring without recognizable cause tend to clear up in a week to ten days, while inflammation caused by infection or chemicals varies widely in course according to the degree of injury.

Treatment.—In most instances the treatment is the same as for eczema. Cleanse and dry the affected parts and apply antiseptic protectives. For local deep inflammations of the limbs, apply an alcoholic sublimate compress until the acute pain and swelling have receded; then follow with equal parts zinc oxide in oil (zinc oil), or zinc ointment, or dusting powder. For rope burns or abrasions, picric acid (3-4 per cent) is excellent. Other useful combinations in dermatitis are:

℞ Zinci sulfatis	ʒ vi	(24 Gm.)
Plumbi	℥ i	(30 Gm.)
Aquae	O i	(500 cc.).

M. Sig. "White Lotion."

℞ Acidi salicylici	℥ ii	(60 Gm.)
Creolini	℥ iv	(120 cc.)
Picis liquidae	℥ iv	(120 cc.)
Sulfuris sublimati	℥ ii	(60 Gm.)
Ol. gossypii	O i	(500 cc.).

M. Sig. For dermatitis.

In the cases of ascorbic acid deficiency treated by Cole et al.,[3] recovery followed the subcutaneous administration of 3 Gm. ascorbic acid.

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LIGHT SENSITIZATION

(Clover Disease—Trifoliosis; Buckwheat Disease—Fagopyrismus; Trefoil Dermatitis; Photosensitization; Bighead in Sheep; Swellhead in Sheep)

Sensitization against light may cause a variety of skin lesions. In general, it is a superficial necrosis of the white markings caused by exposure to the sun after animals have been sensitized to light by eating certain substances, chiefly the legumes. It is not always possible, however, to determine the nature of the sensitizing agent.

Fig. 52.—Paralysis caused by exposure to the sun directly after shearing. (Photograph by courtesy of Dr. Howard Welch, Bozeman, Montana.)

Etiology.—For many years veterinarians have attributed derangements of the skin to the eating of clover—trifoliosis and buckwheat—fagopyrism. It has been found by Hausmann,[1] and Sellards,[2] that certain pigments in the blood, especially hematoporphyrin (iron-free hematin), and artificial fluorescent substances, sensitize living cells to light. In Europe it has been observed that feeding buckwheat is followed by toxic symptoms in animals that are white or have white markings. Koefoed[3] has prepared from buckwheat a fluorescent substance, but according to Sellards "these results do not preclude the possibility that some of the symptoms of buckwheat poisoning are due to anaphylaxis." Toxic effects have been produced in experimental animals by chlorophyll.

In the ambulatory clinic, we occasionally observe superficial gangrene affecting the white parts of the skin of cows at pasture, and to a less

extent this is met with in horses, especially affecting the face. Less frequently one observes a severe affection of the white parts of the limbs of horses pastured on alsike clover (*Trifolium hybridum*, Swedish clover). Swelling and eruption affect the white parts of the lower limbs. Sometimes stomatitis and nervous symptoms are present; several such cases have been described by Fröhner.[4] The disease has been described in Minnesota by Nelson,[5] and in Australia by Bull.[6] Hypersensitiveness was demonstrated in Australia by covering part of the skin with pigment and noting that exposure to the sun was followed by an eruption over the nonprotected area. In a locality in New South Wales, where trefoil dermatitis was common in sheep, Dodd[7] caused erythema, edema, and itching of the skin of guinea pigs by feeding on an exclusive diet of trefoil and exposing them to the sun. Bighead in sheep in Utah and neighboring states was described in 1914 by Frederick.[8] The disease appeared suddenly during spring and early summer, usually after a cold night followed by intense sunshine. Jungherr[9] reports that it is one of the main disease problems in sheep and goats in West Texas; that it is a plant toxemia characterized by jaundice, and necrosis of the liver, and that it may be caused by feeding *Agave lechuguilla* and other range plants. According to Mathews[10] the mortalities in lechuguilla poisoning are due to toxic degeneration of the liver and kidneys. In a group observed by Dr. Welch in Montana, transient paralysis, followed by bighead and superficial skin necrosis on the back, developed in sheep turned out in the sun after shearing on an extremely hot day in June. Phenothiazine may produce photosensitization in pigs, and pigs treated with this chemical should be protected from direct sunlight for at least three days afterward; pigs less that 70 days of age should not be treated with phenothiazine—Swales.[11] In Florida a severe and fatal form occurs in cattle grazed on Bermuda grass on the muck lands of the Everglades.[14]

Symptoms.—In the usual form of superficial necrosis of white areas in the horse and cow, there is first an exudation of serum with matting of the hair, followed by exfoliation of the affected part. Usually there is no other disturbance, though Nelson[5] has described a case in a cow in which there were restlessness, salivation, slight stomatitis, and a temperature of 105° F.

In clover disease, affecting the white parts of the extremities of the horse, as described by Fröhner,[4] there may be icterus, stomatitis, and severe disturbance of the consciousness with paresis. Theiler[12] has described a form of photosensitization in sheep with a mortality of from 25 to 90 per cent. It is characterized by exudation in the cutis and subcutis of the head, succeeded by necrosis of the epidermis, generalized

icterus, and fever. He concluded that the disease was caused by eating *Tribulus terrestris;* it appears after rains succeeded by hot and sunny weather. Rimington and Quin[13] have reported extensively on photosensitization in South Africa; the pigment responsible for the photo-

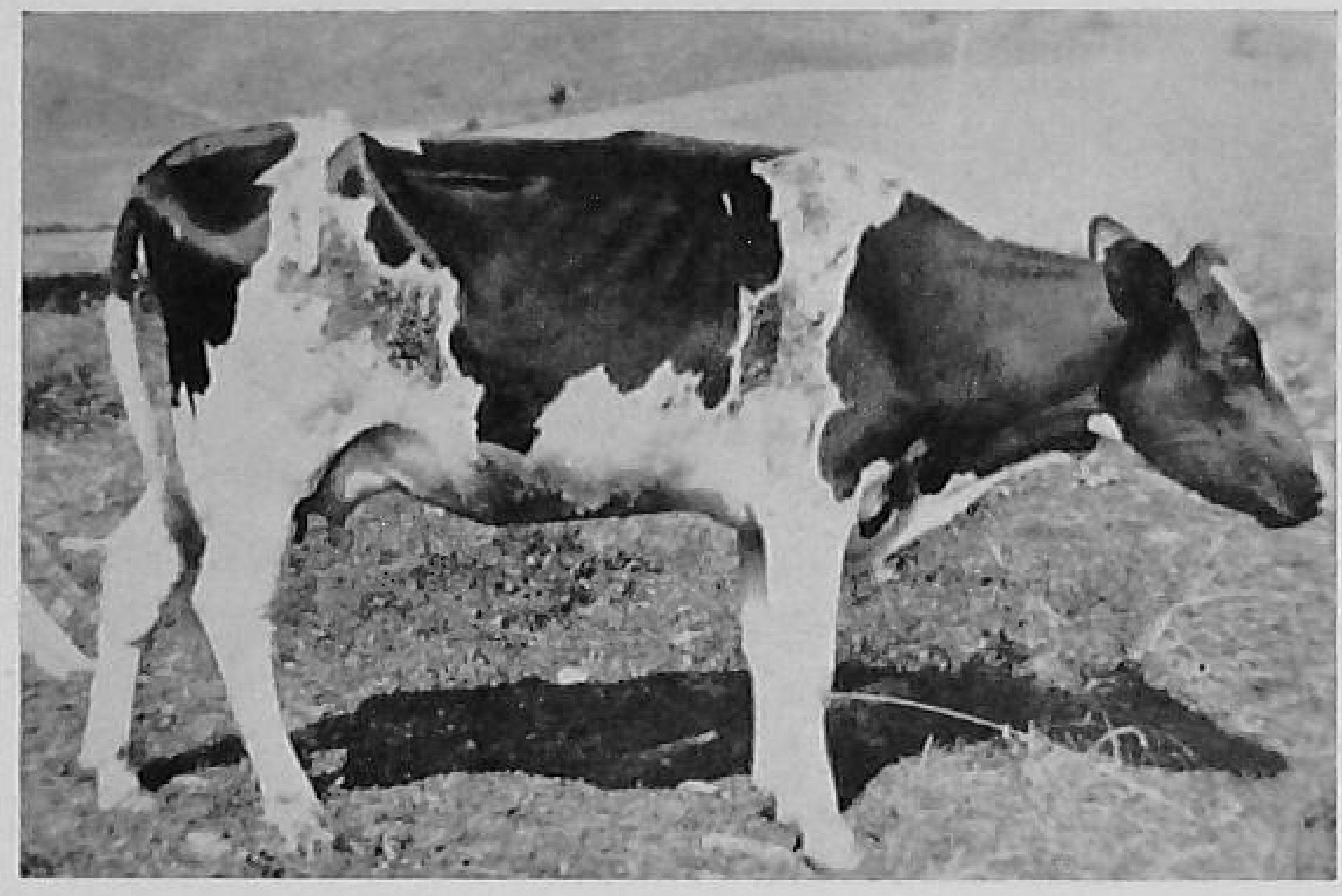

Fig. 53.—Light sensitization. Note superficial sloughing of the white skin over the shoulder and flank.

Fig. 54.—Light sensitization. Superficial necrosis of the white skin on the neck.

sensitivity has been isolated and identified by them as phylloerythrin, a porphyrin derived from chlorophyll.

In the report of the Texas Agricultural Experiment Station for 1931, the symptoms are described as follows: swellhead is characterized by swelling of the soft parts of the head, including the ears, nostrils and throat, which is accompanied in most cases by severe jaundice of the

mucous membranes of the mouth and eyes and of the skin. The course is about 72 hours when the swellings gradually disappear and the skin becomes hard over the affected parts. When placed in the sunlight for several minutes affected sheep will bend down in the back and go down in the hind quarters and finally lie down. The majority die. In New Zealand facial dermatitis causes severe losses in sheep from injury to the liver.

The *diagnosis* of superficial necrosis of the white parts of the skin seems to be difficult to some who have not previously met with the disease. The author has received several letters, enclosing parts of the sloughed skin and asking for a diagnosis of this peculiar affection. In the cow it has been diagnosed as lightning stroke, and in the horse as farcy.

Treatment.—Remove the cause by a change of pasture or roughage. Apply a protective of zinc oxide oil or sulfur iodide and oil to parts that may be congested or inflamed.

According to the reports from the Everglades in Florida,[14] the treatment found to be most effective was sodium thiosulfate, reagent grade, 1 ounce per 100 lb. body weight per vein, or the commercial grade, 2 ounces per 100 lb. orally. There was less than 2 per cent loss in treated animals compared with as high as 20 per cent in untreated.

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GANGRENE OF THE SKIN

Etiology.—In addition to superficial gangrene associated with photosensitization and sudden changes in temperature, certain infectious diseases may be accompanied by necrosis of the skin; among these are hog cholera, swine erysipelas, purpura, and necrobacillosis.

In cows one occasionally meets with primary skin necrosis that closely resembles the form associated with aphthous stomatitis; yet the mouth shows no lesions. The process is gangrenous from the beginning, affecting any part of the body, and, in rare instances, it is generalized. The skin first oozes yellowish serum and then becomes hard and leathery throughout its entire thickness though it may be dry from the beginning. Affected animals usually have a loss of appetite and milk flow and when the necrosis is extensive, depression and toxic symptoms are marked. The course is from one to three weeks.

Decubitis is a special form of skin gangrene caused by prolonged pressure of bruising in animals unable to rise. Chemical agents, such as acids and alkalies, may cause deep injury leading to necrosis. When many animals are confined to a field or yard, as in army camps, infection of the soil may lead to intense and wide-spread gangrene of the feet and limbs.

Treatment.—Decubital injuries from insufficient bedding and neglect are more serious than is commonly realized. In an animal already exhausted, they soon lead to a hopeless condition. It is of first importance to provide a deep bed of straw or hay and to turn the animal not less than four times daily. Other forms of bedding may be used, but fine sawdust, especially when slightly moist, sticks to the moist injured parts and irritates the condition. As protectives, one may employ zinc oxide oil or ointment, or dust heavily with antiseptic powder if the surface is moist. Gangrene of the skin of the limbs responds to moist alcoholic sublimate packs.

ACNE

(Summer Mange; Heat Pox; Sweat Eczema)

Acne is a suppuration of the skin glands with the formation of small nodules or papules. The common pyogenic bacteria, especially the staphylococci, gain entrance to the gland through the influence of friction and dirt at points under the saddle or harness. It occurs chiefly in equines. When horses are exposed to hot weather, sweat, and dust, and are not properly groomed, acne may develop on any part of the body; this is favored by the accumulation of sweat and dirt which close the duct openings.

Symptoms.—Acne most often appears in the saddle region in the form of firm painful nodules, and it may be associated with an eczematous papular or vesicular eruption. In addition to nodules one-half to one inch in diameter which may appear under the saddle, others develop along the back and neck. The presence of nodules at points not under pressure is possibly explained by the spread of infection through the circulation. From the smaller superficial swellings one may press out a small amount of thick pus. Others, especially those not under pressure, may contain a small center of pus revealed only by means of an incision. If pressure and irritation are maintained over the nodules, infection may extend more deeply and result in the formation of boils—*furunculosis.* A common example is the collar bruise, sitfast, on the dorsal surface of the neck.

The course may be acute and associated with a hot painful swelling of the skin in the immediate vicinity, or it may be chronic and recurrent. As a rule the condition is localized but it may spread extensively. When properly treated recovery occurs in from one to three weeks.

Treatment.—The most essential measure is to withhold the animal from work. Arsenical preparations are recommended: Fowler's solution 1 to 2 ounces (30 to 60 Gm.) daily; neoarsphenamine (2 Gm.) intravenously, repeated in four days. For local application, salicylic acid ointment (5 per cent), or alcoholic solutions of disinfectants are indicated: alcoholic sublimate 1:1000 (500 cc.), tannic acid (30 Gm.), and pyoktanin (1 Gm.). Recent acute injury from an ill-fitting saddle may be treated with ice packs for one or two hours, followed by an alcoholic sublimate pack. Nodules may be incised, the contents pressed out and tincture of iodine applied. The antiseptic powder applied for hair root infection may be found useful here: copper sulfate 3 ounces (90 Gm.), powered alum 2 ounces (60 Gm.), tannic acid 2 drams (8 Gm.), and wood charcoal 1 ounce (30 Gm.). A bacterin or vaccine prepared from staphylococci obtained from a nodule sometimes proves to be highly effective.

RINGWORM

(Trichophytosis; Barn Itch; Tinea Tonsurans)

Definition.—An infectious skin disease caused by the fungus *Trichophyton* and characterized by round, sharply circumscribed scabby lesions. These occur chiefly in young cattle in the regions of the ears and eyes.

Etiology.—Among large domestic animals ringworm is largely a disease of cattle, though it may attack the horse and other species. It

often spreads from cattle to man. Calves and yearlings housed in dark, damp stables are most susceptible but it is not uncommon in animals kept under ideal conditions. In certain years it acquires an increased virulence and becomes widespread in a given locality among cattle of all ages. It may spread extensively in pastured young stock, but it is far more prevalent as a stable disease. The seasonal preference is fall and winter, though it may occur at any time. Infection is carried either by direct or indirect contact. The spread from animal to animal is gradual; it is most rapid when several are confined together in a single pen.

Trichophyton.—This is found abundantly in the scabs in the form of threads and round spores. The hair roots are often heavily enclosed in a mass of threads (*mycelia*) and spores. The spores are round and under the microscope they reflect light sharply. Vitality may exist for as long as eighteen months when the crusts are kept dry.

Symptoms.—The period of incubation is about one week. Typical round patches appear around the eyes and ears. These vary in size from 0.5 to 2 inches in diameter and present a gray, dry, crusty surface from which a few broken hairs may protrude. Similar lesions are often found on the neck and they may appear on any part of the body. In certain years, when the disease is especially prevalent, the asbestos-like round lesions develop in many places over the body where hair is black or pigmented. As a rule, however, the condition is confined to a few individuals among the young stock. When neglected it may spread to most of the animals in the herd. In cows the disease may appear in the pelvic region near the base of the tail in the form of bare round hairless spots free from crusts or scabs.

A diagnosis is readily made from the clinical appearance of typical lesions. Microscopic examination of deep scrapings from the margin of a recent eruption reveals the round spores and thread-like mycelia; individual hairs may be surrounded by a thick mantle. In atypical bare lesions the fungus is less readily found, and one may find only spores. It may be helpful to remove the fat by boiling the scrapings in 10 per cent caustic potash solution.

Prognosis.—Ringworm responds promptly to treatment; but in the severe types that spread widely over the body, persistent medication may be required.

Treatment.—The fungus is readily killed by means of disinfectants that penetrate the firm scabs. For this purpose many remedies have been used, but we have found nothing more effective than the following: sulfur iodide (1), light cottonsed oil (Wesson's cooking oil) or olive oil (8-10), and formaldehyde solution to make a strength of 10 per

cent. To prepare sulfur iodide: triturate 20 parts sulfur with 80 parts iodine; place in a bottle and set the bottle in a water bath until fusion occurs. The oil may be added at once in the proportion of 1:8-10; or the bottle may be broken after cooling, the fused mass pulverized, and then combined with the oil. Tincture of iodine may be applied daily. A saturate solution of salicylic acid in alcohol is excellent, especially for ringworm in man. Alcoholic sublimate (1-2 per cent) is said to be highly effective. Where the scabs are thick, antiseptics in oil or ointment penetrate more effectively because of the softening action of the fat. The average case responds promptly to sulfur ointment or sulfur in oil. Other useful antiseptics in this disease are Whitfield's ointment (salicylic acid 1 Gm., benzoic acid 2 Gm., petrolatum 300 Gm.), or picric acid (2 per cent in alcohol). Derris powder 1 lb. (500 Gm.), soap flakes ¼ lb. (125 Gm.), and water 1 gallon (4000 cc.) is effective in the cure of ringworm. Sodium iodide (10 to 15 Gm. in 100 to 250 cc. of water) per vein is also recommended. Dry lime-sulfur 1:40 in water, is useful. Sodium caprylate (mycoban) as a 20 per cent spray repeated every two or three days for five treatments is recommended by Lies.[1]

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MANGE

(Scabies; Itch; Barn Itch; Scab; Acariasis)

Definition.—Mange is a specific contagious disease caused by mites. Prominent symptoms are an eczematous skin eruption associated with itching and loss of hair. The clinical manifestations vary somewhat according to the kind of mite and the species of animal attacked. There are three chief genera of mange mites: *Sarcoptes, Psoroptes,* and *Chorioptes.* There are three chief forms of mange: Sarcoptic mange, caused by burrowing mites, *Sarcoptes scabiei;* psoroptic mange caused by mites which bite the skin and suck blood, but do not burrow, *Psoroptes communis;* and chorioptic mange, caused by mites which live on the surface of the skin but do not bite or burrow or suck the blood, *Chorioptes bovis.*

I. SARCOPTIC AND CHORIOPTIC MANGE

Etiology.—*Sarcoptic mange* has been the most severe, widespread, and important form of scabies. During World War I it was the chief scourge of army horses in Europe. In the United States it is less frequent in horses than in cattle and it is the only form commonly described as affecting horses in North America. In cattle it is rather fre-

quent and it may appear in almost any locality without revealing the source of the original infection. In New York it was reported from six counties in 1925, and from 31 counties in 1946, when it was considered one of the most important health hazards in the state.[3] Imes[1] wrote in 1927 that sarcoptic mange in cattle seemed to be spreading east of the Missouri River, as well as among cattle on the ranges of the West. It is also reported to be increasing in Europe. *Chorioptic mange* was formerly considered to be infrequent and limited to the foot region of the horse and the base of the tail of the cow. In recent years, however, it has regularly been found in association with cattle mange in different parts of the country both alone and in association with the sarcoptic mite. In New York State, within the past three years it has outranked in frequency, injury, and resistance the sarcoptic form, regardless of the fact that the mite neither bites nor burrows. Sarcoptic mange is the most common form in swine in this country, and it is said to be increasing; in 1948 it was reported that more than half of the swine herds in Nebraska and Iowa were infested. In sheep, sarcoptic mange is a relatively mild infrequent form, affecting parts not heavily covered with wool, such as lips, face, and limbs. Camels are attacked in the same manner as the horse and cow. As a rule mange does not spread rapidly from one species to another. Cattle may contact from the horse a mild transient form, which may spread from animal to animal. That it may be transmitted readily between species is shown by a severe outbreak reported in 1915 from West Virginia[2] in which 13 horses, 3 mules, 52 cattle, and 32 people were treated. I have never observed the spread of infection from army horses to soldiers, even when they were engaged in the treatment of mange for months at a time; but I have noted its prompt spread to attendants and students from an occasional mangy horse brought to the college clinic.

The disease is more prevalent in the winter, especially when animals are kept in dark crowded quarters. Yet it may spread extensively in any season among stabled cows, even when care and light are abundant. It is said to extend most rapidly among the young and poorly nourished, but the chief contributing factor is the degree of contact between diseased and clean animals.

The Mite.—Sarcoptes scabiei.—There is a special variety of sarcoptic mite for each species affected; thus we have *Sarcoptes scabiei equi, S. scabiei bovis,* etc. The adult mite is round or slightly oval-shaped, like a horseshoe, with no division between the thorax and abdomen. The length is 0.3-0.5 mm., the color dirty white or yellow. The adults have four pairs of legs supplied with suckers, while the young have three pairs. The insect burrows into the epidermis and the female

lives at the depth of the burrow. Each female lays from 8 to 25 eggs during the egg-laying period, which lasts about two weeks, and at the end of another two weeks the eggs have reached maturity. Mites and eggs live only from two to three weeks after removal from the host. While transmission is usually direct, it is possible to contract mange from infected premises. *Chorioptes bovis* resembles the psoroptic mite in appearance.

Fig. 55.—Sarcoptic mange mite. (Benbrook, Veterinary Practitioner's Bulletin, 1929, 27, 48, Iowa State College, Ames.)

Symptoms.—The symptoms of mange are similar and characteristic in all species of animals. In *cattle* the onset is marked by severe itching around the eyes, face, and neck. In a short time, hairless spots covered with small papules or vesicles, appear. Dandruff is usually abundant. In bulls the early eruption may be acquired from service, when it develops on the inner surfaces of the thighs or along the abdomen. After a few weeks the skin becomes wrinkled in thick folds, loss of hair is extensive, and an eczematous eruption may spread over the entire body. In the *horse* mange usually starts on the head, neck, and shoulders, but it may first appear on the body, flanks, or thighs. Intense itching causes the horse to rub; the skin becomes bare in irregular patches; and exam-

ination reveals an eruption of vesicles and papules. The eruption spreads rapidly or slowly, according to the general condition of the animal and the care of the skin; it may extend over the body in two or three weeks. In a short time the hair is filled with bran-like dandruff and in places it may be matted together with moisture. Finally, the loss of hair is general, the skin becomes thickened, wrinkled in thick folds, and covered with crusts and scabs. It may become leathery and hard and ooze blood where the scabs are detached, especially over the hips. The

Fig. 56.—Sarcoptic mange.

leathery condition may remain for weeks after the mites have been destroyed. Under crowded stable or paddock conditions, all animals in the group are attacked, and the disease soon becomes serious unless treated.

In *swine*, mange caused by *S. scabiei suis* may start around the eyes, nose or ears, though it shows a preference for the legs and lower part of the body. The general appearance is like that of mange in other species. It spreads most rapidly under close confinement, and is said not to spread among healthy hogs kept outside in the sun during the summer.

Diagnosis.—Mange may be positively confirmed by microscopic examination of deep scrapings from a recent lesion. Boil the scrapings in a 5 per cent solution of sodium hydroxide and examine under a low-power microscope. Presence of the eggs or the mite is conclusive. In animals that have been treated, repeated examinations may fail to reveal either eggs or mites in scrapings from advanced cases of mange. In such circumstances one is justified in a positive diagnosis by the

contagious character of the affection, the itching, and the characteristic skin eruption. The persistent use of fly repellents may cause an eruption on the horse similar to that of mange. No mites are found. The hairless condition over various parts of the body develops more rapidly than in mange, and at a season of the year when mange is usually inactive. The eruption of an extensive acute eczema appears suddenly over a large part of the skin, while that of mange first appears in certain spots and gradually spreads. Hyperkeratosis has been diagnosed

Fig. 57.—Sarcoptic mange.

as mange, and Dr. Baker[3] writes that "in several herds cases of persistent pruritus and scabbiness have occurred in which no etiologic agent could be demonstrated."

Treatment.—The remedies formerly used in the treatment of mange in cattle have been replaced by benzene hydrochloride (BHC) containing 99 per cent gamma isomer ("lindane"). As used in the New York State program and recommended by Cornell University[4] for mange control, it consists of 1½ pounds of a 25-per cent lindane wettable powder per 100 gallons of water or a 0.046-per cent lindane spray. Two applications are applied within an interval of a week or ten days. The

equipment should be a hydraulic sprayer capable of developing at least 350 pounds pressure, and delivering 7-10 gallons a minute. The operator must take great care to cover every part of the animal's body including the face, and he should use at least two gallons of spray per animal. Nozzle equipment has consisted of a five foot rod usually made of ½ inch galvanized pipe with two livestock type nozzles placed in line, one at the end of the rod and one about eight inches from the end and delivering at right angles to the rod. The cut-off most convenient for this purpose is the pistol grip type cut-off. There should be a swivel in the hose line where it attaches to the rod so that the rod can be easily turned to any angle.

"It has been found that the addition of 10 pounds of wettable sulphur to the above lindane dosage gave somewhat better results than the lindane alone. While lindane usually gives control for a period of three or four months, the lindane wettable sulphur combination appeared to give all season control, whether or not it accomplished eradication will not be known until examinations can be made about a year after the treatment.

"While it is reported in the literature that very young calves may be killed with lindane dosages as low as 0.5, many hundreds of calves have been treated in New York with a 0.046 dosage without illness or death occurring in any known case."

"The fact that many of the herds which were treated early in the winter, and which appeared to be free of the disease during the first quarter of the following year but showed evidence of infection in the latter part of March and April, indicates that our present system of treatment will not eradicate the disease. There is practically unanimous agreement that the treatment was worthwhile, since it prevented the development of the usual mange outbreak in late winter, and also protected the people handling the animals from contact infection."[3]

"Continued examination of animals sprayed in 1950 showed that the only herd in which two sprayings had effected a permanent cure was sprayed both times with 0.075 per cent concentration."[5]

The use of chlordane on milk-producing dairy cows is prohibited by the U.S. Food and Drug Administration.

Treatment of Mange in Swine.—In 1948 Spencer[6] reported success in the treatment of sarcoptic mange in swine with one application of chlordane. This was used in a 0.25 per cent chlordane solution, made by putting 500 cc. of 74 per cent emulsifiable concentrate into 50 gallons of cold water. It was applied with a power sprayer using from one to two quarts per head with 50 to 150 pounds pressure. The tests indicated that one application of a 0.25 per cent chlordane solution, when thor-

oughly applied, will completely clean up sarcoptic swine mange. "The use of lindane in the proportion of 4 pounds of the wettable product (containing about 0.25 per cent of the active ingredient) per 100 gallons of water is an effective means of destroying the mites on hogs."[7] "All stages of swine mange are completely eradicated by one thorough application of BHC spray that contains 0.13 or 0.15 or 0.26 per cent gamma isomer."[8]

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II. PSOROPTIC MANGE

(Sheep Scab; Cattle Scab; Common Mange)

Etiology.—The most prevalent form of psoroptic mange in the United States is in *sheep,* and this seems to be the only form of mange in sheep in this country. Mange formerly caused heavy loss in the sheep-raising districts of the West, and numerous government bulletins on the subject have been written.[1] In 1920 Hall[2] reported that "quarantine and eradicative dippings have cleaned it out of a greater part of the United States, so that at present it is largely a matter of cleaning up the relatively small amount that is scattered about, an exceedingly difficult task, however, because of the scattered condition of the infection." Occasionally, scab-infected sheep from the West carry mange to individual herds in the State of New York. In the Winter of 1930 a badly infected flock was found in Tompkins County, and in the Winter of 1943 several counties in the western part of New York State were quarantined because of widespread sheep scab.

In 1952 Newsom[3] wrote that "due to activity of the state and federal sanitary officers and the enforcement of dipping regulations, it was almost eliminated. During the last two or three years, however, it has flared up again in some of the southern and midwestern states."

In *cattle* the history of psoroptic mange is much like that of sheep. Among range cattle in the western part of the United States it has been

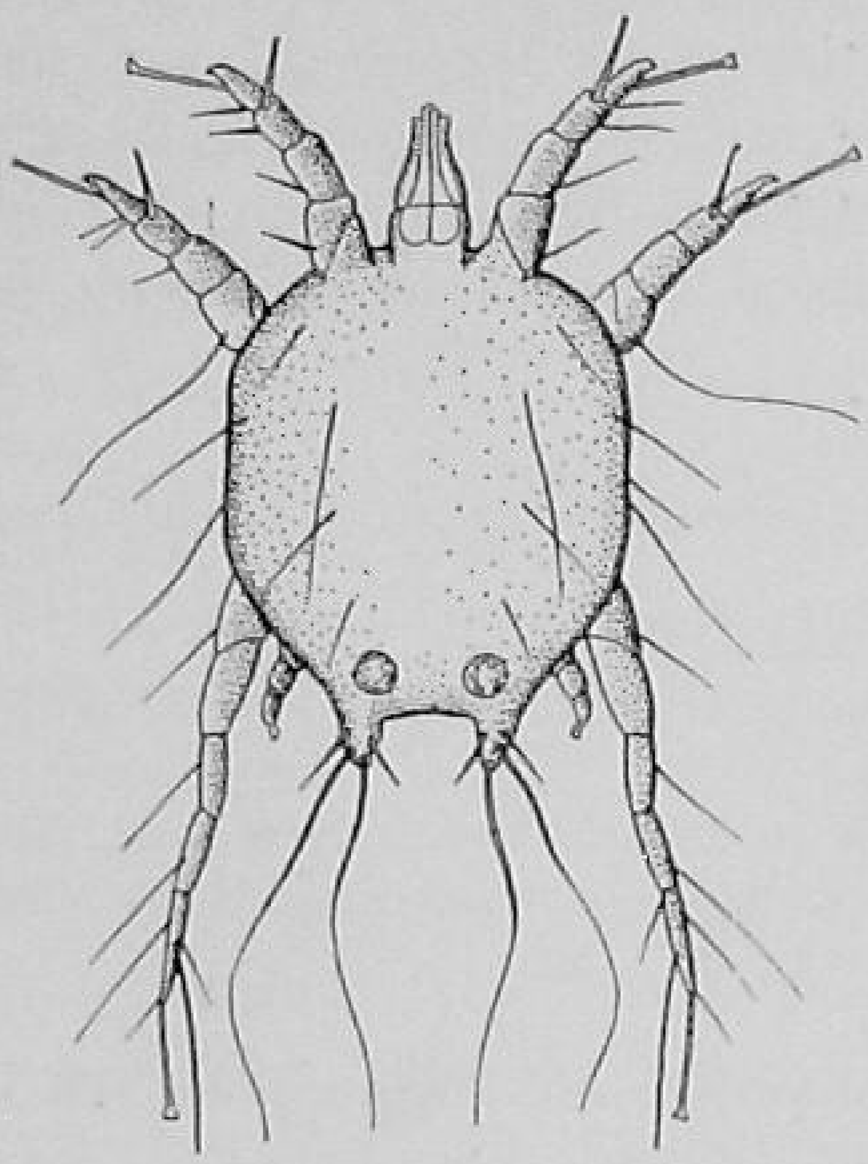

Fig. 58.—Psoroptic mange mite. (Fitch, An. Rep. New York State Vet. Col., 1916-17.)

a serious disease, but it has been greatly reduced by means of dipping and quarantine, and is probably limited to scattered areas in the West. In 1942 psoroptic mange mites from two herds of cows in the vicinity of Ithaca, New York, were identified by Dr. Baker.[4] Psoroptic mange in *horses* is said to occur in this country, but I have not found a recorded observation of a single case. It is improbable, however, that horses in the western states have entirely escaped it. It is stated in B. A. I. *Cir. 148*[5] that the disease is more highly contagious to all classes of horses than sarcoptic mange; that each species of animal has its own variety of psoroptic mange; and that the variety which lives on the horse, ass, or mule is not transmissible to other animals, with the possible exception of the camel. *Swine* are not attacked by the psoroptic mite.

The Mite.—*Psoroptes communis,* var. *ovis.*—The adult psoroptic mite (sucking mite) has an oval body 0.5 to 0.8 microns long. It is white or yellowish in color and when placed on a black background it may be seen with the naked eye or a hand-glass. The head is elongated and pointed, and both the head and four pairs of legs extend much farther behind the body than in *Sarcoptes.* The entire life cycle is passed on the host. Eggs are laid on the skin and hatch in from 1 to 5 days. Within 10 to 12 days after hatching the young mites reach maturity and deposit eggs. The adults live from 30 to 40 days on the host, and not more than 10 days when kept in moist jars at room temperature. The psoroptic mite penetrates the epidermis, sucks lymph, and sets up a small inflammatory reaction with exudation of serum and the formation of crusts. Among sheep the disease is highly contagious and spreads almost entirely by direct contact; it is not transmissible to other animals except goats. Infected corrals left empty were found free from infection after 17 days (Mönnig).

Symptoms.—In *sheep,* mangy areas usually first appear on the back and sides. The mites puncture the skin to suck the blood, thus causing irritation, infection, and an eczematous eruption. This leads to itching,

rubbing, and biting. Finally, crusts and scabs form and the wool becomes loose and falls out, leaving bare areas. Neglected flocks are emaciated and the mortality is high. After mange is introduced into a clean flock, one or two years may elapse before it becomes generally distributed in an advanced form. In a severely infected flock, most of the individuals will be found rubbing against posts and fences, biting themselves, and pulling out wool. In addition to extensive bare areas

Fig. 59.—Psoroptic mange.

on the back and sides, a scabby condition of the skin will be found around the eyes, head, and neck. Yet symptoms may appear within a week to ten days after exposure of healthy individuals. Thirty days is the commonly accepted incubation period. It develops more rapidly in fine wooled breeds.

In *cattle* the first lesions appear on the withers or around the base of the tail, from whence they spread over the back and sides. As in sheep, the pricking of the skin by the mite causes itching and rubbing with the formation of an eczematous eruption; this finally results in loss of hair, wrinkling of the skin, and extensive formation of crusts and scabs. Death from disease of the skin and emaciation may result. In the *horse*, psoroptic mange first appears on the protected long-haired parts of the skin—under the mane, at the base of the tail, and in the intermaxillary region.

Diagnosis is readily made by an examination of scrapings from recently infected areas.

Treatment.—As in other forms of mange, benzene hexachloride has replaced all previous remedies. A single dipping in an unheated aqueous suspension of technical benzene hexachloride (BHC) containing 0.06 gamma isomer eliminated scabies from more than 3600 sheep, including lambs 1 to 5 weeks old, in 4 bands on New Mexico ranges.[6] The efficiency of this treatment has also been reported by Kemper,[7] and by Kemper and Roberts.[8]

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III. DEMODECTIC MANGE

(Follicular Mange)

Demodectic or follicular mange occurs in swine, cattle, and horses, but it is infrequent and of little importance in these species. It has been reported from 14 States, chiefly in old cows, where it is a cause of damage to leather. The lesions are in the form of nodules ranging in size from that of a pinhead to a pea or hazelnut. Finally the nodules break and discharge a thick creamy pus. In cattle, they most often appear in the skin of the neck, shoulders, and chest. As described in the B.A.I. Report for 1940, it was not experimentally transmitted by direct or indirect exposure and none of the attempts to cure the disease were successful. In swine the nodules first appear on the face and spread over the under side of the body and inner sides of the legs. In cattle, they are said to appear first on the shoulders and neck. The condition is incurable, but the nodules may be few in number and cause no serious trouble.

SPINOSE EAR TICKS

These ticks are prevalent in the Southwestern States in large numbers in the ears of cattle, to a less extent in those of horses and dogs. They cause extreme irritation manifested by shaking and rubbing the head

and rolling the body. The United States Bureau of Animal Industry[1] recommends as particularly effective both as a lethal and a repellent agent, a mixture of benzene hexachloride (12 per cent gamma isomer) 5 parts, xylol 10 parts, and pure pine oil 85 parts. A mixture of chlordane 5 parts and pure pine oil 95 parts, introduced into the ear by the use of a spring-bottom, pint size oiler, was equally effective. These remedies provide partial protection for at least a month.

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PEDICULOSIS

(Lousiness)

There are two main groups of lice affecting domestic animals—sucking lice and biting lice. Each species of animal has its own species of lice. Of the two forms suctorial lice are the most injurious.

Cattle harbor three common species of lice: *Haematopinus eurysternus* (short-nosed suctorial "blue louse"), found chiefly on mature animals; *Haematopinus vituli* (long-nosed suctorial "blue louse"), found chiefly on calves and young stock; and *Trichodectes scalaris* (biting "red louse"). *Horses* also harbor three species: *Haematopinus asini*, of chief importance, and the biting lice *Trichodectes pilosus* and *T. parumphilosus*. *Swine* are affected by only one species: *Haematopinus suis*. This is the largest louse affecting domestic animals and it may attain a length of one-fourth inch. *Sheep* are chiefly infested with the sucking louse *Haematopinus ovillus*, as well as the biting louse *Trichodectes sphaerocephalus*.

Sucking lice are recognized by their large size, pointed heads, and bluish bodies. The biting louse is much smaller, the head is broad and round, the color of the body is yellowish white, while the head is reddish. The entire life history of lice is passed on the animal. Eggs ("nits") are attached to the hair near the skin where they hatch in about two weeks, and two weeks later the young females begin laying eggs. Lice do not survive more than a week when separated from the animal, but under favorable conditions, eggs upon detached hairs may continue to hatch for two or three weeks.

Symptoms.—The presence of lice upon animals is almost universal, but the number depends largely on the nutrition and the extent to which an owner will tolerate vermin. They are most abundant and breed most rapidly in the winter in long-haired poor stock. With the coming of spring, when the hair sheds and the animals go to pasture, lousiness is greatly diminished. The favorite locations for sucking lice are the

head, the sides of the neck, the back, and the inner surface of the thighs. Biting lice may be found anywhere, but the favorite locations are the withers and base of the tail. Irritation caused by lousiness retards growth and nutrition. Loss of hair and eruption of the skin may appear, as in mange and ringworm. Rubbing and licking and restlessness are common.

Treatment and Control.—Thorough grooming is an effective remedy against lice. In the treatment of individual cases, the animal may be clipped and given a thorough bath with creolin solution (4-5 per cent) or any of the coal tar cresote dips. In suitable weather, preferably in the fall when the lice are not abundant, and a number of individuals are to be treated, a hand spray pump or an orchard spraying outfit may be used for the application of the antiseptic. Since the eggs are not destroyed, it is advisable to repeat the treatment in 16 days to destroy the newly hatched lice. Animals should be watched for a time after the second application to determine whether a third treatment may not be required. Nicotine sulfate solution (5 cc. Black Leaf 40 to one gallon of water) destroys lice on contact when applied as a spray, according to Roberts and Legg[1] of Australia. Raw linseed oil is effective, and it can be applied in cold weather. One pint is enough for a single treatment for four or five cows; apply with a stiff brush (*Storrs Ag. Exp. Sta. Bul.* 97, 1918). Derris powder is effective against lice. In the B.A.I. Report for 1941 it is reported that "one treatment with washes and sprays containing one pound of derris powder to 100 gallons of water, and derris powder mixed with various inert powders in the proportion of 10 per cent of derris powder, and applied dry, killed all lice on the treated animals but did not destroy the viability of the eggs. Two treatments 16 to 17 days apart eradicated the lice."

DDT, 0.25 per cent solution, or 10 per cent powder, is highly effective against lice. Since the residual DDT in the hair coat will also kill newly hatched lice, only a single treatment is necessary. A dip containing 0.75 per cent DDT, tested on 300 heavily infested pigs, not only killed all motor lice within four hours, but continued to kill newly hatched lice thereafter, and effectively eradicated *Haematopinus suis* from the herd —Kemper and Roberts.[2] Aqueous suspensions of wettable lindane containing as little as 0.06 per cent of the active ingredient, when used as sprays on cattle heavily infested with short-nosed and long-nosed sucking lice, apparently destroyed all lice in 24 hours. Swine dipped in a vat containing benzene hexachloride suspension containing 0.06 gamma isomer were permanently free from lice.[3] Lice on sheep were effectively destroyed by dipping in an aqueous suspension of wettable lindane containing 0.045 per cent of the active ingredient.[3]

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WARBLE FLIES OF CATTLE

(Botfly; Breeze-fly; Ox-Bot; Gadfly; Cattle Grubs; Myiasis)

Infestation with larvæ of warble flies is reported to be the cause of rather extensive losses among cattle in North America and Europe. Australia and South Africa are said to be free of warble flies. In Southern Europe, in Denmark, and Sweden, damage from larvae has led to extensive control measures, sponsored and supported in part by public funds. Hadwen[1] reports it is estimated in Canada that 27.5 per cent of hides are injured yearly; from the tanner's point of view the warble season is from January to early in July. The prevalence of flies varies widely in different parts of the country, and it also varies widely in different years. If the fly season is wet and unfavorable to oviposition, the number of bots will show a definite decrease the following spring. In herds where infestation is heavy, it is a serious annoyance and the cause of considerable loss.

In 1922 Shannon[2] reported, "Within recent years it has been noticed that the botfly injury to cattle and horses, particularly in certain portions of the country, has been greatly increased. This is due to the unintentional importation of additional species of European botflies which were quite unknown in America a little while ago.

"The most striking characteristic of the increased botfly injury to cattle is seen in the great number of 'running' cattle during June and July, which results in decreased milk supply, and not infrequently in serious accidents to the animals. The so-called European ox-warble, *Hypoderma bovis,* DeGeer, is responsible for this change. The first authentic finding of this species in the United States was in 1910—at present this species is one of the chief cattle pests in the region where it occurs—New York, New England, and Canada."

Life History.—Two species of cattle gadflies are widely prevalent. *H. bovis* and *H. lineatum.* The flies vary in their seasonal occurrence and in their method of depositing eggs, but the migration of the larvae is essentially identical for both species.

H. bovis (Bomb Fly) is 14 mm. in length; it has yellow hair on the anterior part of the thorax, and dark-brown wing veins; ventral surface

of abdomen and thorax are nearly black; tail end, orange yellow; legs are clean, with few hairs. The flies appear in June and are active until the first of August. Oviposition is on the outside of the thighs and on the legs above the fetlocks. The insect flies at about the level of the stifle joint, striking twenty or thirty times rapidly; it then leaves for a few minutes and returns to repeat the attack. The eggs are deposited singly and cemented to the root. All animals are terrified when a fly begins an attack.

H. lineatum is 12.7 mm. long; ventral surface of thorax, black; wing veins nearly black; reddish-orange tail; legs rough and hairy. The flies appear about April 15, and are active for about six weeks. The favorite places for oviposition are the hair on the cow's heels, fetlocks, knees, and hocks; when the cow is resting the eggs are deposited at the line of contact between the body and the soil. The fly alights gently and lays a number of eggs in sequence on a hair. Oviposition excites only range animals.

After about a week, the eggs hatch, the larvae immediately penetrate the skin through the hair follicles and begin their migration which terminates the following spring when mature larvae appear beneath the skin of the back. In this migration it is believed that the larvae move chiefly in the loose connective tissue of the subcutis; they do not enter the circulation or the muscles. In December they are chiefly found in the walls of the esophagus. In March they have migrated from here to the subcutis of the back. According to Hadwen,[1] they leave the esophagus at its junction with the rumen, pass through the neural canal, and arrive in the subcutis, where they bore through the skin. The time required for this final migration is apparently short. After the larvae emerge from the skin and drop on the ground, the time required for the pupal stage is about thirty days, when they emerge as mature flies.

The *symptoms* observed in association with the development of the larvae within the body consist: (1) in a period of frenzy when the fly deposits eggs; (2) in an occasional skin eruption when large numbers of larvae are penetrating the skin, *hypodermal rash* of Hadwen; and (3) various degrees of inflammatory reaction caused by the presence of the mature larvae beneath the skin of the back. Under usual conditions, heavy invasion may result in unthriftiness and lessened milk flow, and occasionally it may lead to extensive abscess formation. Hadwen and Bruce[3] have described an anaphylaxis of cattle and sheep produced by larvae. Their natural cases among cattle present symptoms which are practically identical with those observed by us in urticaria, and the fact that about 60 per cent of our cases of urticaria in cattle appear in the months of March to May suggests that they may be examples of

Hypoderma anaphylaxis. It has been observed that larvae are most numerous in young cattle and other animals kept constantly at pasture.

Control.—The only known effective method of control consists in the destruction of the larvae after they arrive beneath the skin in the winter and spring. This method is successful because of the limited flying range of the adult fly. Experiments conducted by Davies and Jones[4] in Wales, who treated cows in various demonstration areas, led to marked decrease in the infestation. They attribute their success to the very local habit of the fly. Similar observations have been made in Denmark and Sweden. Bishopp[5] and associates conclude that the results of control measures applied by an individual cattle owner when he is more or less surrounded by livestock are likely to be disappointing; they have observed a number of instances, however, when well-isolated dairymen and breeders have largely, if not completely, eradicated the pest from their herds by systematic destruction of the grubs.

For the destruction of larvae beneath the skin derris root has proved to be 100 per cent effective. As described by Schwartz,[6] The Federal Bureau of Entomology and Plant Quarantine recommends the following mixture for use as a spray: derris powder or cube root (5 per cent rotenone content) 5 pounds; wettable sulphur, 10 pounds; water, 100 gallons. The mixture so made up is adequate for treating 200 head of cattle. Powders containing equal parts of derris cube (5 per cent rotenone content) and wettable sulphur may be dusted from a shaker on the backs of cattle with one hand and worked into the hair with the other hand. When so used these powders are very efficient larvicides, and one pound of the mixture is sufficient for 36 animals. A wash made up of adding 12 ounces of derris powder or cube powder (4 to 5 per cent rotenone content) and 2 to 4 ounces of neutral soap to each gallon of water, is a reliable treatment when applied with a sponge, cloth, or brush. . . . A more convenient way to apply the wash is with a home-made sprinkler which consists of a fruit jar with holes punched in the metal lid. As the liquid flows from the jar it is spread with a stiff, fiber brush into the hair coat and skin of the back and upper sides of the animal. . . . The spraying method is especially useful in the treatment of range cattle and must be applied with a power sprayer capable of developing about 250 pounds of pressure. Dusts act more slowly on grubs than washes or dips.

To destroy all of successive arrivals of larvae beneath the skin, four treatments during the season of emergence are required. In the latitude of the State of New York the larvae first appear in the subcutis soon after February 1, and they begin to emerge from the skin about March 15. It is recommended, therefore, that the treatment begin March 1

and be repeated three times at intervals of not more than 35 days. The percentage of effectiveness of single treatments averaged about 90 per cent in observations described in the Federal B.A.I. Report, 1944, p. 37.

Among other preparations reported by Bishopp[5] as highly effective in the destruction of larvae are: derris powder, 1 part, petrolatum, 10 parts; rubbed in, one application. Silver nitrate, 5 per cent solution injected with a syringe. Nicotine dust, 2 per cent dusted into the hole. Pyrethrum flowers, 2 pounds; alcohol (denatured, formula 5), 1 gallon; inject with an oil can. Carbon tetrachloride injected with an oil can.

Mechanical removal through the openings in the skin is widely practiced in Denmark. The larvae are first pulled partly through the opening with a small needle curved in the shape of a crochet-hook, when they are grasped with a pincette and withdrawn. Removal by squeezing or other mechanical means is most effective if done early before the larvae approach maturity. Extensive abscess formation often develops at the seat of mature larvae, causing the cow great discomfort, as well as loss in condition and milk. Once an abscess has formed, it is impossible to expel the larvae by the usual method of squeezing, or hooking with a curved needle.

In the U.S.B.A.I.[7] program for destruction of cattle grubs, the cattle are sprayed with a mixture consisting of 7½ pounds of cube powder (5 per cent rotenone content) per 100 gallons of water, applied at 400-pound nozzle pressure. . . . From 1945 through 1948, 308 cattle were treated by hand only once during the grub season with the standard rotenone wash. The average number of grubs was reduced from 17 in 1945 to 3.5 in 1948, as a result of these treatments. . . . When hand washes and sprays were correctly applied, they were about equally effective. . . . Jet sprays, applying a solid stream to the backs of the cattle under high pressure, were far more effective than the usual cone sprays. . . . The correct time of treatment may vary from year to year by as much as six weeks.

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DISORDERS OF METABOLISM

AZOTURIA

(Hemoglobinemia Paralytica; Lumbago; Myoglobinuria; Lumbar Paralysis; Black-Water)

Definition.—A specific disease of horses, affecting chiefly draft animals, characterized by a suddenly developing paralysis of the hind limbs, which appears during exercise after a brief period of idleness. Associated symptoms are profuse sweating and dark or black urine (myoglobinuria). The chief anatomical change is degeneration of the iliopsoas and quadriceps femoris groups of muscles.

Etiology.—Azoturia is a frequently fatal disease wherever draft animals are used, and it is not rare among the lighter breeds. With few exceptions it occurs in the months of October to May. The disease is met with only in well-nourished animals. When such individuals, at regular work, are kept idle with no exercise and no reduction in diet for from two to five days, an attack may develop in from fifteen minutes to an hour after exercise is resumed. This predisposing condition is the most essential causative influence. An attack is unusual after a rest period as brief as one day or as long as two weeks. The intensity of the exercise or work is of little significance; the paralysis develops even when the horse is being led at a walk. Infrequently an attack may develop when the horse is in the stable, without exercise. Carlstrom[1] believes that a storage of glycogen in the muscles increases the formation of lactic acid which causes muscular exhaustion, circulatory obstruction and deranged motility.

Morbid Anatomy.—The chief changes are found in the iliopsoas and quadriceps groups of muscles. On section, these present areas of pale and friable muscle similar in general appearance to that of fish. On exposure to air the light-colored areas become somewhat red. Immediately after death the reaction of affected muscles is acid. Histological examination shows a waxy degeneration of the entire muscle, including extensive areas that present a normal macroscopic appearance. To a less extent there are cloudy swelling and fatty degeneration. Inflammatory changes are almost completely absent. In severe attacks, degenerative changes of the muscles may be general, even involving the muscles of the heart. Roman and Martin,[2] on the other hand, state that "besides the extensive degenerative changes in the contractile sub-

stance of the muscle, a very definite inflammatory process has been found, a myositis."

Next to the muscles, the most marked changes are found in the kidneys. The presence of hemoglobin in the kidney may be recognized macroscopically in sufficiently severe cases by the presence of dirty-brown spots on the cortex and of streaks in the marrow substance. The kidneys may be slightly enlarged. Histologically there are found deposits of hemoglobin and marked fatty degeneration in the epithelial cells. In mild attacks there may be no kidney changes.

Symptoms.—Within one-half hour after leaving the stable, sweating begins, the gait becomes stiff, and the animal is reluctant to move. Usually the lameness effects one or both hind legs, but occasionally it may be confined to a front leg. If given complete rest at the onset of the attack, the symptoms may disappear within a few hours, and the case go on to complete recovery. More often the symptoms continue, even with prompt and complete rest. Unsuccessful efforts to remain standing finally lead to a dog-sitting position, and soon the victim goes down, lying flat on the side. These efforts are accompanied by restlessness and signs of marked abdominal pain. The animal makes repeated unsuccessful efforts to rise, and sweating increases until the horse is completely bathed in moisture. Localization in the muscles of the front leg is less serious; usually the horse is able to remain standing. In some cases the attack is both front and rear.

On palpation the affected muscles, psoas, quadriceps femoris, are found to be hard, and there may be an edematous swelling of the affected region. Such swellings are more frequent in affections of the front limbs. The gluteal muscles show increased tension, but the hard, board-like consistency of the diseased groups is lacking. There is no psychic disturbance. The temperature is about normal, though it may be elevated late in the course of a severe attack. In severe forms the pulse is rapid, irregular, and weak. The respiration is fast at the onset and this increase continues throughout the course in severe forms. The mucous membranes are congested, and in severe attacks icteric.

The appetite may be normal at first if the pain and restlessness are not too marked. As complications develop, the appetite is lost. Thirst is normal. Peristalsis is diminished, though there may be repeated defecation at the onset.

Urination is somewhat difficult, and the urine may be normal, increased, or diminished in amount. The color is bright red, reddish brown, or very dark brown, almost black; in rare instances the urine may be normal in a severe attack. The urinary sediment contains hemoglobin cylinders, kidney epithelium, and some red cells, though

the latter two may be absent. According to Carlstrom[1] the dark or black color of the urine is caused by products of degeneration of the muscle, and not by hemoglobinuria.

The course is widely variable. In a few hours recovery may be complete, or the horse may be dead. The prognosis is good if the animal remains standing, or is able to stand with the aid of slings. Recoveries usually occur in from two to four days. If the hind limbs are unable to support the weight within the first few days, the prognosis is unfavorable. Death comes from paralysis of the heart due to myocarditis, from uremia induced by the kidney lesions, or from decubital septicemia. Chronic muscular atrophy may leave the animal in a worthless condition. Unless the predisposing causes are repeated, there is no tendency to recurrence.

Treatment.—The first requirement is to provide absolute rest. If possible, keep the animal in a standing position; if this is impossible, provide heavy bedding to prevent decubital abrasions. Cover with blankets, and bolster up on the sternum with parts of bales of hay or other similar material. If the animal is very restless, delirious, or apparently suffering from pain, give a narcotic. Prompt effect may be had from chloral hydrate administered in solution through a stomach tube. If a rectal examination reveals distension of the bladder with urine, catheterization may give relief. There has been much debate over the question of artificial emptying of the bladder, but there is no doubt concerning the relief obtained by the patient when this overdistended organ is emptied. It is desirable to turn the animal at least once every four hours, but it is seldom possible to obtain the requisite assistance. The condition of the bladder should be watched in order to prevent painful distension. Practically all authorities advise the administration of a laxative. For this purpose, mineral oil 2 to 4 quarts (2000-4000 cc.) seems preferable to aloin, because of its lack of irritative action, and the tendency of an aloin purge to induce weakness. Whatever the nature of the disease may be, it is associated with irritation and weakness and anything that contributes to these two conditions should be avoided. For this reason do not administer arecaline, or other drugs which have a similar action. Supply a reasonable amount of hay and grain and water frequently. In severe cases, for which there is little hope at best, constant attendance is essential. Otherwise the patient, in an attack of delirium or restlessness, may drag itself a considerable distance, destroy all chances of recovery, and become hopelessly cast. The continuous application of moist hot applications over the affected muscles should assist the circulation through these parts. All authors recommend such applications. Often they are applied over

the gluteal region only, leaving untreated the psoas and rectus femoris group where the degenerative changes are most frequently located.

Numerous remedies have been administered for their alleged beneficial effect upon the muscular and other changes, but there is little evidence that any of them are beneficial. To overcome the effects of acid substances which enter the circulation, alkaline preparations have been given. For this purpose Dieckerhoff introduced bicarbonate of soda. At the present time this is given intravenously; Hertha[3] recommends 5 liters of a 2 per cent solution prepared just before use. Since Dieckerhoff advanced the theory that in azoturia the blood stream receives large amounts of acid substances from the muscles, oxidation and alkalization of the tissues have been practiced. It was Dieckerhoff who introduced the use of bicarbonate of soda (150-300 grams) combined with sodium sulfate (300-500 grams) on the first day, followed by daily doses of 50-200 gm. bicarbonate of soda. The use of similar substances has been stimulated by the conclusions of Hertha,[3] Carlström,[1] and others that the essential cause of the disease is an excessive increase of lactic acid in muscles overladen with glycogen. Numerous medicinal remedies for azorturia have been recommended, and none has proved to be of any definite value. A number of reports have appeared on the favorable effect of calcium gluconate, but its value in this disease remains to be established. Cardiotonics and epinephrine (adrenalin, ephedrine) have been employed in conjunction with sodium bicarbonate.

A few recoveries following the injection of insulin have been reported by Wright[5] but the numbers are too few to indicate its real worth. It has been given in a dosage of 100 to 200 units daily, both subcutaneously and intravenously. Wirth-Diernhofer[4] quote a report that the mortality has been greatly reduced by the use of an analgesic (novalgin) given in the vein and muscle, combined with calcium gluconate 80 Gm. and boric acid 15 Gm. in a liter of water per vein, and cold applications.

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PARTURIENT PARESIS

(Milk Fever)

Definition.—Parturient paresis is a paralysis and loss of consciousness leading to coma in dairy cows that have recently calved; often the onset is marked by tonic muscular spasms and increased excitability of the peripheral nerves (tetany). There is a sudden drop in the blood calcium—hypocalcemia, which is thought to be the cause. Since the parathyroid glands apparently control calcium metabolism, attempts have been made to associate the phenomena of milk fever with disturbed function of these glands. There is an increase of blood sugar—hyperglycemia.

Etiology.—Because of the frequency of milk fever, and the heavy losses therefrom in high-producing dairy cows, it has for years been the subject of intensive study. Little information concerning its control was obtained until Schmidt,[1] a practicing veterinarian in Kolding, Denmark, introduced mammary injection of a solution of potassium iodide, in 1897, on the theory that the disease was caused by toxins formed in an actively secreting udder, that iodide of potash would reduce the secretions, and indirectly the formation of toxins. Through a relatively large milk catheter, a rubber tube and a glass funnel, he injected 5 grams of iodide of potash in 0.75 of a liter of freshly boiled water. He comments that with each injection the column of air in the tube was forced into the udder, and since the funnel was often empty this occurred repeatedly. In conclusion he wrote that infusion of iodide of potash in the udder, especially in combination with atmospheric air, and followed by massage, was in most patients followed by a typical course which often led to rapid recovery. Of 50 cases treated in this manner by Schmidt, 46 recovered, two died and two were slaughtered. He writes that he was less impressed by the recovery, than by the unusual rapid recovery of so large a percentage of very sick patients. Finally, a combined report of the treatment of 412 cases of milk fever by 65 veterinarians, registered a cure in 90 per cent. It is not clear from this report, however, that the action of air was fully comprehended. But to the practicing veterinarian in Kolding, Denmark, with his discussion of over 50 pages on milk fever, belongs the credit for one of the most important discoveries in veterinary medicine. Finally, Anderson,[2] of Skanderborg, discovered that inflation of the udder with air alone was highly successful. Thus it was demonstrated that mechanical distension of the udder effected a prompt cure, while the cause of the disease remained a mystery.

Calcium Deficiency.—The generally accepted theory as to the cause

and essential nature of milk fever attributes it to an *acute calcium deficiency*. This hypothesis was advanced by Dryerre and Greig[3] in 1925. In explanation of this theory Greig[4] writes, "The colostrum of the cow is rich in calcium, and it was considered likely that the onset of a profuse lactation might occasion a rapid reduction in the concentration of the blood calcium. This idea seemed to be supported by our further observation that the spastic seizures which often characterize the early stages of milk fever were tetanic in character. We believed that the mere mechanical withdrawal of calcium from the blood as the result of the onset of a profuse secretion of milk could not in itself be regarded as the cause of milk fever, because if this were so, every heavy milking cow would be subject to the disease. For that reason we postulated that some other factor, therefore, must act as a predisposing cause, and we suggested that such might be found in parathyroid dysfunction." But no proof of a parathyroid dysfunction in milk fever has been presented.

In support of this theory, the author[4] demonstrated the following facts:

"There is no difference between the calcium values in parturient cows and those in non-parturient cows and bullocks.

"The onset of milk secretion is accompanied by a transient but appreciable fall in the blood calcium, which returns to normal after the crisis of initiation of lactation is passed.

"In milk fever there is invariably a pronounced fall in the blood calcium. The degree of severity of the symptoms bears a distinct relation to the calcium level in the blood. From a series of observations made in one case, before and during the attack, the fall in calcium appears to be abrupt; it is coincident with the onset and corresponds with the progressive severity of the symptoms.

"In an examination of 81 cases of diseased conditions in cattle other than milk fever, none was found to present a hypocalcemia in any way comparable with that which maintains in that disease.

"Inflation of the mammae of normal lactating ewes causes a rise in the blood calcium (about 10 per cent).

"Inflation of the mammae of the cow in cases of milk fever results in a pronounced rise in the blood calcium. The rise is at first rapid, and the case usually shows definite signs of recovery when a level of about 6 to 7 mg. of calcium per cent has been reached.

"Injection of calcium gluconate, exclusive of other treatment, elicits specific curative response in milk fever.

"The subcutaneous injection of calcium gluconate can abort the milk fever attack. Evidence is submitted that the calcium injection imme-

diately after calving and preferably reinforced by a second injection about 24 hours later would prove a preventive treatment."

Experimenting with lambing sickness in sheep, the identity of this disease with milk fever in cows, and the curative effect of calcium gluconate, were established.

In milk fever in cows the blood calcium drops from a normal of 10 mg. per 100 cc. of serum to a minimum of 3.0 mg. per 100 cc. and a maximum of 7.76 mg. per 100 cc.

On the basis of this evidence, and the rapidly curative effect of calcium gluconate, there is general acceptance of the opinion that the essential cause of milk fever is an acute blood calcium deficiency.

In explanation of the action of inflation of the udder, Greig[4] advances the theory "that the curative effect is a mechanical one in that it elicits mammary distension and so prevents the further interchange of calcium from the blood to the gland acini. There is also reason to believe that the calcium, which is heavily concentrated in the gland, is forced back into the blood as the result of the mammary distension."

Greig[5] states that half a gallon of colostral milk contains as much calcium as exists in the blood at any one time. This suggests that the lack of calcium in the blood in milk fever is not a genuine deficiency, but a disturbance of the calcium distribution.

The view expressed by Barker,[6] that "milk fever is seen to be a much more complicated disease than a simple hypocalcemia," is sustained by a report on 300 cases comprising milk fever, acetonemia, grass tetany, transit tetany and pregnancy toxemias. Thus a hypocalcemia of parturition may be linked up with any one of the following: (*a*) hypermagnesaemia, (*b*) normal magnesia value, or (*c*) hypomagnesaemia. He reports that "clinically one is able to distinguish these three associations by studying the behavior of the animal: (*a*) where a hypocalcaemia is linked with hypermagnesaemia the patient reels, becomes paretic and narcosis supervenes; (*b*) where a hypocalcaemia is linked with a normal magnesium the patient 'paddles' with the hind legs, becomes recumbent, may or may not get up, finally becomes paretic and coma supervenes; (*c*) where the hypocalcaemia is linked with a hypomagnesaemia the patient shows tetany of the fore end, as well as the hind limbs, hyperaesthesia, and may become recumbent followed by a convulsion." He also reports a lowered inorganic phosphorus in the majority of cases showing tetany.

The increasing frequency of milk fever, and of deaths or relapse following the usual treatment, suggest that the commonly accepted theories of its cause and nature are still incomplete.

The age of onset is at the height of milk production. Of 113 cases

treated in our ambulatory clinic in three years, only 1, a 4-year-old, was less than 5 years of age, and over 90 per cent were between the ages of 5 to 9 years; 48 were recorded as either 7 or 8 years old. Approximately 25 per cent also suffered from some other puerperal disease, chiefly retained placenta. Four died: one on arrival, two of pneumonia, and one of torsion of the uterus. In a series of 77 cases of milk fever in one large herd, five were recorded at the second parturition; the youngest was 3 years and 11 months old and the average for the five was 4 years and 2 months. The seasonal incidence in this herd, surveyed by Henderson,[7] was 4.07 per cent of susceptible parturitions in the months of May to September and 13.35 per cent in the months of October to April. Metzger and Morrison[8] reported the highest seasonal incidence in the months of January to April. In the ambulatory clinic the milk fever incidence is highest from September to May, when the number of parturitions is highest, and in the same month there may be twice as many cases in one year as there are in another. The incidence of milk fever according to breed is highest among the Jerseys. In the series of 77 cases compiled by Henderson[7] from a large herd under the care of the ambulatory clinic, the percentage according to breed was as follows:

Jersey	29.2	Guernsey	8.6
Brown Swiss	15.3	Ayrshire	6.0
Shorthorn	13.3	Holstein	5.6

These observations are similar to those reported by Metzger and Morrison.[8] Certain individuals suffer from milk fever at each parturition, and such recurrent attacks are most prevalent among Jerseys.

Morbid Anatomy.—The postmortem examination is negative. Anatomical changes characteristic of milk fever have not been observed, though the uterus shows little or no involution. Cows that die after intermittent improvement and relapse may show marked degeneration of the liver which is yellow and friable.

Symptoms.—The onset of the attack is usually within twelve to seventy-two hours after birth of the calf. An attack previous to expulsion of the fetus is not rare, and it may occur months later. Because of the variety of abnormal conditions in which the milk fever syndrome may develop, and respond to treatment, the diagnosis in attacks remote from the time of parturition needs confirmation by a blood test for calcium; in the few cases observed in our clinic there has been a hypocalcemia. At first the cow is depressed and not inclined to move, symptoms which represent a beginning loss of consciousness. Occasionally there is a brief initial period of excitement, muscular twitching,

hypersensitiveness and convulsive movements of the head and limbs, which soon are replaced by the more characteristic paralysis and depressed consciousness. In its most typical form, the paralysis first affects the hind limbs. Difficulty in standing is soon followed by recumbency, often with the head resting on one side, and a gradual passing to complete coma. According to Greig,[4] the initial hypersensitiveness and convulsive movements are genuine tetany. In diseases characterized by loss of consciousness, the period of depression may be preceded by one of excitement, so that the exact interpretation of this particular syndrome may be in doubt. Whether it is tetany of parathyroid dysfunction, or an initial excitement incidental to loss of consciousness, is not clear.

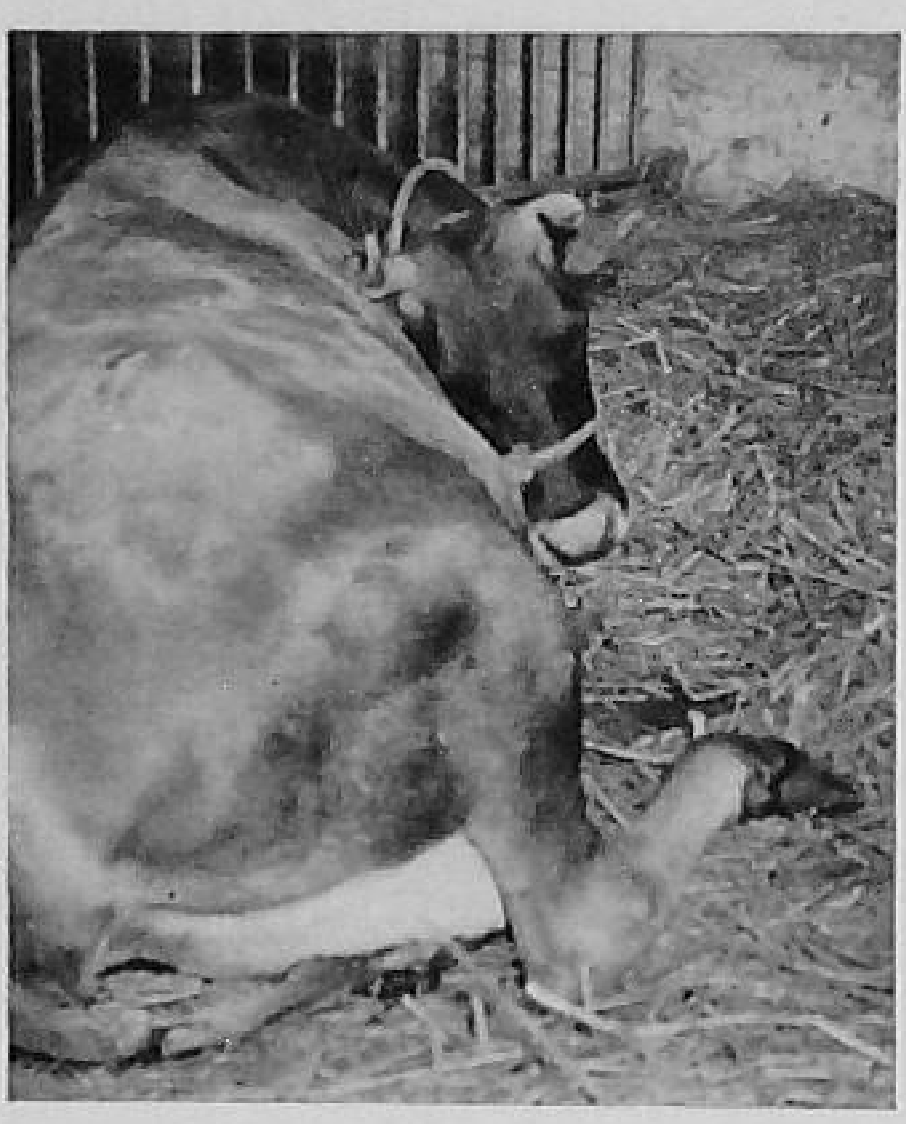

Fig. 60.—"Typical" milk fever attitude. Seen in about 20 per cent of cases. Less frequent than "kink" of the neck. (Courtesy of W. J. Gibbons.)

In the majority of cases, the dominant symptoms are paresis and depressed consciousness, without signs of motor irritation in the form of twitching, clonic spasms, or other involuntary movements. One of the most constant and characteristic signs is a tonic spasm of the muscles of the neck, giving it a distinct lateral kink. Often the cow is found down and unable to rise. The eyes have a dull or staring expression, the pupils are dilated, and often the conjunctival mucosa is congested. There is a complete loss of appetite, and failure to eat may be the first symptom to be observed by the caretaker. The muzzle tends to be dry, and the horns, teats, and extremities are cool. The pulse is from 50 to 85, and the temperature from 97° to 101° F. When milk fever is associated with febrile disease, as pneumonia or heat stroke, the temperature is high. Groaning is sometimes present, while the respirations are frequent, shallow, and labored according to the intensity of the attack. Drooping of the ears is frequently present. There is an atony of the digestive system which causes suppression of the bowel evacuations, and in common with other paralytic conditions the anus is relaxed. Tympany is frequent in recumbent cases. When coma is advanced, and the body in lateral position, the contents of the rumen may regurgitate and be inhaled, causing fatal pneumonia.

In the case reports by Schmidt,[1] he frequently mentions distention of the bladder with urine, kinking of the neck, and failure to react when the pupil of the eye is touched. In milk fever the pupil is dilated, and there may be drooling of saliva.

The *blood* has been examined chemically by numerous research workers in an effort to discover some explanation of the cause and nature of the disease. In the interpretation of changes in the blood, one needs to consider that practically all abnormal conditions found in the blood are secondary. Hayden[9] was the first to demonstrate that a condition of hyperglycemia exists in milk fever, while Fish[10] observed a decrease in the phosphates. The work of Greig,[4] which revealed a decrease in calcium, has previously been mentioned.

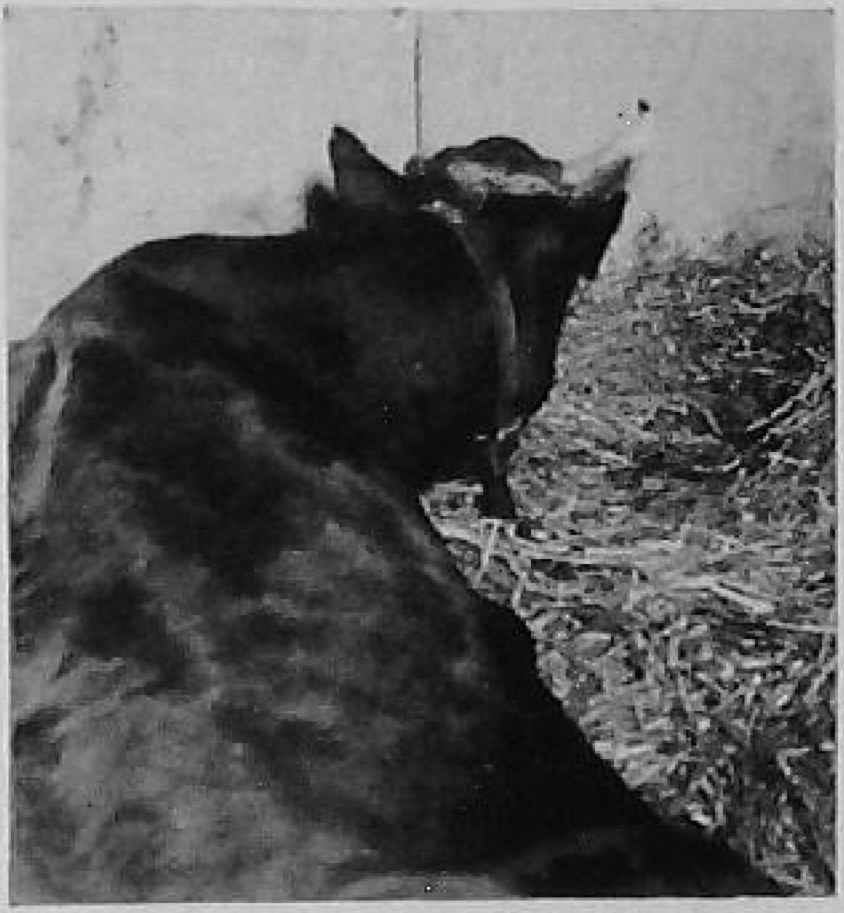

Fig. 61.—Lateral kink of the neck, characteristic of milk fever. (Courtesy of W. J. Gibbons.)

The *course* in untreated individuals is from a few hours to a few days, usually ending in death. Under proper care and treatment the mortality is not over 3 or 4 per cent, and is usually the result of complications, such as inhalation pneumonia, or associated diseases, as dystocia, torsion of the uterus, enteritis, etc. In one case at pasture the cow suffered fatal injuries from rolling down a hill. There is always the possibility that a severe attack may end fatally before treatment is available.

Diagnosis.—Uncomplicated typical attacks of milk fever are easily diagnosed. Yet it frequently happens that atypical symptoms, and a milk fever syndrome caused by other affections, such as septic mastitis, make a positive diagnosis difficult at first. Metritis is a frequent complication. The patient may present typical milk fever symptoms, and fail to respond to treatment. Because of the size of the uterus, and the possible absence of exudate in the vagina, an immediate diagnosis may not be possible. When the disease fails to respond to treatment, or responds only temporarily, and the paralysis persists for twenty-four hours or more, a careful examination of the uterus should be made. When such ainmals are unable to rise, an examination of the uterus may be unsatisfactory. To determine the contents of the uterus, one may pass a rubber catheter, inject a small amount of solution and

siphon back to determine the presence of pus. Even this may fail, the return flow being clear when the uterus contains pus. When death occurs regardless of the milk fever treatment, it may be the result of metritis. For this reason one cannot make a diagnosis in fatal cases, except by means of a postmortem examination.

Evidence that many cases of so-called atypical milk fever are associated with acetonemia, makes it necessary to consider the essential syndrome of this disease. Acetonemia may occur in younger animals. In general, an examination of the urine or blood will reveal a marked increase in the total ketone bodies. There are cases, however, in which laboratory examination may reveal characteristics of both diseases. For example, in a cow attacked eight days after parturition, examination of the blood gave calcium 5.70 (hypocalcemia); sugar 34.84 (hypoglycemia); and total acetone 11.10 (acetonemia).

When milk-fever-like symptoms appear near the end of pregnancy, one needs to consider whether it is a case of milk fever or of septic metritis in a sealed uterus (it may be both) or mastitis. Even after the case responds to milk fever treatment, there may be a prolonged attack of metritis with various degrees of paralysis.

When milk-fever-like symptoms appear several months after parturition in association with acute mastitis, or indigestion, and disappear promptly after inflation of the udder or the administration of calcium, the diagnosis may not be entirely clear. It may be either milk fever or acetonemia; usually it is milk fever.

When the onset is especially violent and the cow kicks and groans and froths at the mouth and rolls and lunges about, and has a wild expression, there is a tendency to suspect meningitis, possibly from lead poisoning. But when such cases respond to inflation of the udder or administration of calcium the diagnosis of milk fever or acetonemia becomes established. Whenever any acute disease appears at the time of parturition, it should be regarded as a parturient disease until there is positive proof that it is otherwise.

The *prognosis* is good in uncomplicated cases. When the onset precedes the act of birth, or occurs within six to eight hours thereafter, prompt treatment is especially desirable; there is a tendency for such cases to take a rapidly fatal course, and relapses are not infrequent. When milk fever is intercurrent with dystocia, prolapse of the uterus, or severe metritis, there is a similar tendency to fail to respond to treatment, and to relapse after temporary improvement. We have records of three cases of combined milk fever and prolapse of the uterus, each of which ended fatally.

Treatment.—Since the discovery that inflation of the udder with

air results in a rapid cure in practically all uncomplicated attacks, the air treatment has been in universal use. This treatment immediately brought under control one of the most destructive cattle scourges of the dairy industry. Probably there is no other example of so prompt a transformation from impending death by so simple a remedy.

Following the discovery of calcium deficiency by Dryerre and Greig,[3, 4] the administration of calcium was suggested. This, also, has proved to be rapidly effective as a cure. In typical uncomplicated cases, recovery may occur in as short a time as ten or fifteen minutes after its introduction into the blood stream. A dosage of 375 cc. of a 20 per cent solution of calcium gluconate,* intravenously, plus 125 cc. subcutaneously, is usually followed by prompt improvement. To avoid the danger of heart failure from too rapid administration the intravenous injection should be made through a small needle, not over 16-gauge in size, and over a period of 15 to 30 minutes. The subcutaneous injections may be distributed in several places. When administration is too rapid the pulse may drop to 30 and become irregular; when given slowly there is usually no change in the frequency of the pulse. In case of slow response or recurrence, this dosage may be repeated within three or four hours. It is a common practice to give 1000 cc. of a 20 per cent solution at the first dose, and to administer as high as 2500 cc. over a period of three days. Because of the frequency of acetonemia in association with milk fever, veterinarians often give an equal amount of 40 per cent dextrose solution mixed with the calcium gluconate solution, and it is believed by some that this dilution of the calcium adds to its safety. In the presence of acetonemia the chief danger to the patient still is from hypocalcemia. In areas where a deficiency of magnesium is suspected, as well as in cases that fail to respond to calcium-dextrose treatment, a combination of calcium, magnesium, and dextrose may be useful; if response is still delayed, inflation of the udder with air is indicated. There are various reports that magnesium sulfate, ¼ pound orally to cows with a tendency to relapse, may be followed by prompt recovery. When recovery is slow, or the attack is recurrent, there is a tendency to supplement, or even replace, calcium gluconate injections with some

* Calcium gluconate	1600 grams
Boric acid	320 grams
Aquae q.s.	8000 cc.

Bring water to boiling, add boric acid, continue to boil and add the calcium gluconate. Heat for 5 to 10 minutes, filter through cotton and gauze while hot into sterile bottles. Add 3 drops (plus) of phenol to each bottle. Wash bottles thoroughly and rinse with water at 180° F. Boil rubber stoppers and funnels, stopper and cool gradually. (Use 95-98% phenol solution.)

stimulant such as Amfetasul. In the treatment of such cases, however, one needs to consider that no remedy except calcium, or inflation of the udder, or a combination of both, has been found to cure hypocalcemia. Whenever a supplementary stimulant, such as the widely used amphetamine sulfate (Amfetasul, 3 to 5 cc.) is administered, it should either precede or follow other medication by an interval of at least ten minutes. In areas where magnesium deficiency is known to occur, injections of magnesium sulfate, or oral administration of 4 ounces (120 Gm.) may be beneficial.

A frequent complication in milk fever is failure of the patient to attempt to rise following recovery. The following method may be successful: tie a hame strap around each hind ankle, connect with short links, carry a rope forward from the connecting links, tie to the base of the stanchion, push the feet under the cow, and stimulate with an electric torch.

In some cases, recovery is slow, and remissions occur, regardless of the treatment used. When inflation with air proves unsatisfactory, the use of calcium may lead to prompt recovery; and when calcium fails, inflation with air may effect a cure. In some instances, recovery is slow even when both treatments are applied. In obstinate cases the use of dextrose (500 cc. of a 40 per cent solution) per vein is sometimes beneficial. Cushing[11] has described a case of milk fever in which he inflated the udder six times over a four-day period, the cow being down and up five times; following the last inflation she received dextrose (120 Gm. in 500 cc. of water) per vein. In the absence of sufficient data on blood analysis to explain recurrent attacks, the most plausible theory is a recurrence of the hypocalcemia. And this condition is met by larger and more frequent dosage of calcium gluconate solutions, half of which may be given subcutaneously because of more prolonged resorption. So long as symptoms persist, if no more than knuckling of the fetlocks, calcium gluconate should be continued.

In discussing the treatment of milk fever, Barker[6] states that "the subcutaneous injection of a 25 per cent solution of magnesium sulfate seems to be the therapeutic find of the last few years. As an adjunct to the calcium boro-gluconate solution or inflation, and injected at an independent site, it hastens the restoration to normal in the mixed hypocalcaemia-hypomagnesaemias, and is often the deciding factor in recovery where the hypomagnesaemia is pronounced. Injections of 200 cc. may be repeated at short intervals with impunity in all cases of hypomagnesaemic tetany wherever occurring in the life history of the patient. The licking mania type of case carrying acetonemia responds at once to magnesium therapy." Cases of combined hypo-

magnesemia and hypocalcemia have been described by Muth and Haag[12] in Oregon.

The advantages of udder inflation are simplicity of operation and low cost. A disadvantage is the possibility of infection of the udder, causing mastitis. The prevalence of the disease is at an age when mastitis also is most prevalent. If mastitis already exits, it cannot be detected because of the distended condition of the udder. Thus an inflation performed with the greatest precaution against infection may be followed by an inflammation. It is also possible that inflation may introduce infection from which chronic inflammation develops at a somewhat later period. This source of infection has been suspected in cows with a history of milk fever.

Equipment for the treatment of milk fever must be constantly in readiness, for there is usually not sufficient time for proper disinfection of utensils after the case is on hand. The teat catheter should be boiled and wrapped in sterile material with whatever inflation apparatus may be used. First, wash the udder and teats thoroughly with warm soap and water, followed with a warm disinfectant solution. This cannot be done properly unless the cow is in a clean roomy place, supplied with clean bedding, and the udder resting on clean cloth, as a folded sheet or towel. Inject each quarter until it is fully distended, and tie a piece of bandage around each teat to retain the air. The bandages should be removed after three or four hours to prevent pressure necrosis. If the cow fails to respond, a second distension may be made after six to eight hours. There is little danger of over-inflation, but after the ligatures are removed there may be a relapse from escape of air. For an inflation apparatus I prefer a rubber bulb with a metal cylinder filled with cotton. This equipment is compact; it can be wrapped in a sterile towel and constantly carried as a part of the regular equipment.

In the general care of milk fever patients, the cow should be kept on the sternum by means of sacks filled with straw and packed along the side. Lying flat on the side may result in regurgitation of the contents of the rumen and fatal pneumonia. Avoid drenching. In the days before the inflation treatment, many cows were lost through drenches that went to the lungs because of paralysis of the throat.

As a prophylactic, Greig and Dryerre[3,4] recommend an injection of calcium gluconate immediately after calving. Delay in milking, and incomplete milking, have been practiced for the prevention of milk fever. This precaution is apparently successful in reducing the number of attacks. The effect is to maintain pressure within the udder, similar to that induced by inflation. Because of the possibility that it may cause mastitis, or aggravate one already present, it is not recommended.

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MILK FEVER IN THE GOAT, EWE, AND SWINE

Small ruminants and swine are subject to attacks of milk fever, but the disease in this country is relatively rare. In his work on calcium in the blood, Greig examined five ewes affected with "Lambing Sickness" and found a marked decrease in the calcium. He reports that affected animals respond with amazing rapidity to air inflation, and that in one case a subcutaneous injection of calcium gluconate brought about rapid recovery. The symptoms in goats are identical with those observed in the cow; the onset may precede expulsion of the fetus, and prompt recovery follows udder inflation.

In *sheep* the disease is also known as "Milk Disease." According to the description by de Bruin (Holland) milk fever ("milk disease") in sheep may occur just before or shortly after lambing, but more often the onset is six weeks later, one or two days after the lamb is weaned.

In *swine* the onset is shortly after parturition, as in the cow. The symptoms are inappetence, diminished milk secretion, and long recumbent periods. Fever is usually absent, though in hot weather the temperature may be high. Recovery follows injection of calcium gluconate (100 to 150 cc.) into the muscles or the peritoneal cavity. Hand-feed the

pigs with the following: 1 pint milk; 1/4 cup cream, unless Jersey milk is used; 1 tablespoonful brown sugar; and 1/2 cup limewater. Warm to body heat and give each pig 2 tablespoonfuls every 4 hours; or put the milk in a shallow pan and the pigs will soon drink it.

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KETOSIS IN COWS

(Acetonemia; Acetonuria; Ketonuria; Hypoglycemia)

Definition.—Ketosis is a disturbance of carbohydrate metabolism leading to an intoxication from an excess of ketone bodies in the blood. It may be caused by underfeeding or overfeeding (primary); it is commonly secondary to parturition, less frequently to other conditions. It is identified clinically by depression, loss in condition, anorexia, incoordination of movements, lethargy, and less often excitement. Blood and urine examinations reveal an excess of ketones (ketonemia, ketonuria), and the blood shows a marked decrease in glucose (hypoglycemia).

In 1849 Landel[1] wrote a description of a peculiar disease in a cow which probably was a case of what is now termed the nervous form of ketosis. The patient was a 6-year-old Swiss cow first seen September 2, eight days after normal calving. She had broken loose, stood with her head against the wall, was slobbering, and grinding her teeth. When seen by Landel she was greatly excited and the eyes were staring. There soon developed genuine catalepsy with loss of consciouness and sensation; from this she recovered under treatment by bleeding, producing blisters, and placing cool packs. But the symptoms returned. Standing with her head pressed against the manger there were marked slobbering, grinding of the teeth, protruded tongue, and finally complete catalepsy. These attacks alternated with improvement until she at last made a complete recovery. This condition he termed *mania puerperalis* in contrast to *febris puerperalis*. Fleming's Obstetrics[2] contains an excellent description of the symptoms of *mania puerperalis* which is marked by "great agitation, sometimes fury." For treatment chloral hydrate was recommended and chloral hydrate is still highly regarded by veterinarians of wide experience.

Ketosis has been chiefly reported from Holland, Denmark, Sweden, and England. Either it is less frequent in other countries, or it has been unrecognized. It has been reported from Texas by Hayes,[3] and from Mississippi by Alston.[4] The latter reports "palsy after calving" to be the most common disease of recently freshened cows in his practice. In Germany it was described in 1908 by Janssen.[5] In 1928 Hupka[6] described

cases in the vicinity of Hanover, and expressed the belief that the disease is frequent in Germany, though it has rarely been recognized.

When the first edition of this book was written in 1933, the work of Sampson and Hayden[7] had shown that some of the cases diagnosed as atypical milk fever in our ambulatory clinic were actually ketosis. After the development by Hayden of a test for acetone in the urine suitable for use in the stable, it was soon recognized that ketosis is very prevalent, both as a primary clinical condition and in association with other diseases; and that an excessive amount of acetone in the urine, acetonuria, is not infrequent in cows that are apparently normal. In the year ending July 1, 1940, 75 cases of ketosis in cows were treated in the ambulatory clinic of the New York State Veterinary College, and in the year ending July 1, 1950, there were 284 cases with 6 deaths. In some herds ketosis is a serious problem, while in others, kept under apparently indentical conditions, it is infrequent.

Etiology.—In explanation of the variable and somewhat obscure etiology of ketosis, the classification by Hoflund[8] as primary acetonemia due to underfeeding or overfeeding, and secondary acetonemia which occurs especially just preceding calving or when a cow ceases to eat because of some disease, is useful.

The chief form follows overfeeding at calving time when usually there is a change from a cellulose-rich dry ration to an added amount of high-protein milk ration. A cow that produces 30 quarts of milk per day loses 3 pounds of sugar, and the main part of the sugar must be produced by the rumen through cellulose fermentation. When this fermentation is retarded by an abrupt increase in the high protein feed a part of the sugar requirements are obtained from fat; this results in an augmented flow of fat to the liver where it is changed into sugar and releases acetone bodies which appear in the blood. Under high feeding acetonemia may be provoked by addition of molasses to the diet. Underfeeding acetonemia, on the other hand, may be prevented or cured by feeding molasses. This type occurs in cattle on poor pastures or insufficient roughage or poor roughage, in areas where farming is backward.

At the time of parturition, the cow is not only exposed to the strain of high milk production and change in diet, but also to a variety of secondary factors, such as retained placenta, metritis, and mastitis.

The fate of glucose in the metabolic system and its relationship to the utilization of the ketone bodies has received a great deal of attention in recent years. It has been known for a long time that glucose plays an important oxidative function in providing energy for work. Recent work summarized by Peters,[9] indicates that glucose has an important operative function in metabolism which is to provide an intermediate com-

pound required for the proper function of the metabolic steps by which acetate and ketone bodies from fat are metabolized to carbon dioxide and water, and the energy released for work such as milk production. In the absence of adequate amounts of glucose arriving at the tissues, as is the case in acetonemia, acetate derived from fat is inefficiently oxidized and accumulates in the system as ketone bodies. The animal, apparently in an attempt to combat this deficiency, uses amino acids from tissue protein to provide the essential intermediate compound, but the overall process is less efficient than where glucose is available so that the blood ketone level remains high, and rapid emaciation from the metabolism of body protein results.

Acetonemia affects cows of all ages; in our series the ages appearing most often are two, five, and seven, but each age from two to ten has been represented. It occurs in each month of the year, and in both stabled and pastured animals. In a series of 226 cases of acetonemia treated in the ambulatory clinic of the New York State Veterinary College over a three-year period from July 1943 to June 1946, the monthly distribution was as follows: June, 4; July, 5; August, 9; September, 10; October, 14; November, 19; December, 28; January, 26; February, 19; March, 34; April, 33; May, 25. Many of these were in association with other affections, but the majority were uncomplicated and all were diagnosed and treated for acetonemia.

Hendershot,[10] in the Hawaiian Islands, reports its presence in grade Herefords of good quality and of all ages and sexes under range conditions on a diet of Kiawe bean. Samson[12], on the other hand, writes that so far as he is aware no one has presented evidence showing that significant ketonemia occurs in steers, bulls, wethers and rams irrespective of the disease with which they may be affected.

In routine examinations of entire herds of high-producing well-fed cows, ketones are found in the blood and urine of many apparently normal animals. This indication of low carbohydrate is explained by Shaw[11] and others as a depletion of the liver glycogen reserve due to an increase in utilization of glucose by the udder at parturition. Shaw concludes that "the development of ketosis is due to faulty carbohydrate metabolism at a time when the demands for carbohydrate are greatest and in which the inability to maintain a normal level of glycogen plays a major role." Shaw observed that the liver glycogen was low prior to parturition and the onset of ketosis; that the mammary gland continues to remove a normal quantity of glucose from the blood, even in marked hypoglycemia; that regardless of normal food intake the milk production is adversely affected; that carbohydrate deficiency is not the cause, since hypoglycemia is not

prevented by liberal feeding of molasses prior to parturition; and that liver damage may account for the failure of many cases to respond readily to glucose therapy. There is no impairment of the capacity of the various body tissues to utilize glucose. In both normal cows and those approaching ketosis, the blood glucose may drop before parturition, and even on an adequate diet it may remain low for five or six weeks.

The frequency of persistent acetonemia in many highly fed high-producing herds is presumably due to deranged carbohydrate metabolism; while in ordinary herds where the disease is less frequent the deficiency may be due to a poor ration. An inherited tendency is suspected by a few whose observations have included three or more generations in purebred herds.

Morbid Anatomy.—The mortality is low, and cows that have died of uncomplicated ketosis reveal extreme fatty infiltration of the liver[12] and kidneys, a condition like that of pregnancy disease in ewes. The subject is usually a high-producing cow with a history of ketosis in previous lactations.

Symptoms.—Ketosis may be divided into two chief clinical groups: parturient and nonparturient. In a series of 100 cases 50 per cent were parturient and approximately one-half of the nonparturient occurred within thirty days after parturition. At least three clinical types, which may overlap somewhat, are recognized: the milk-fever or paresis syndrome; the digestive syndrome; and the nervous form manifested by excitation, delirium, motor irritation and paresthesia. The first two include the great majority. The parturient group occurs chiefly within two days of parturition and usually follows it; about two-thirds are associated with the milk-fever syndrome and one-third with various affections of the uterus; occasionally the digestive or nervous type is parturient. In the nonparturient group about two thirds present the digestive syndrome, while the other third is about equally distributed between the nervous type and those in which increased acetone in the urine is symptomatic of other diseases, such as pneumonia and traumatic gastritis. In the nonparturient group the milk-fever syndrome is infrequent.

The digestive type of acetonemia occurs in well-fed heavy milkers in from ten days to six or more weeks after parturition. There is a sudden or gradual loss of appetite, a rapid decrease in condition, and usually a marked decrease in the milk flow. While the onset is usually sudden, there may be a history of unthriftiness or a gradual loss in condition or milk production over a period of from one to four weeks. It is possible that most cases develop slowly and become clinically

distinct only when the glycogen has been reduced to a certain level. The cow stands with the back arched, the head lowered, and the eyes half closed; often the eyelids twitch. The temperature is usually normal, but in exceptional cases it may be as high as 107° F. The pulse is variable and seldom exceeds 80. The breathing is normal, though it may be rapid or slow and labored. Atony of the rumen and scanty evacuations are the rule, but diarrhea is not unusual. The physical signs may be limited to a loss in condition and milk flow, or the cow may become thin in flesh and continue to milk heavily. A few have wandered about the pasture, and staggered and knuckled at the fetlock and fallen down.

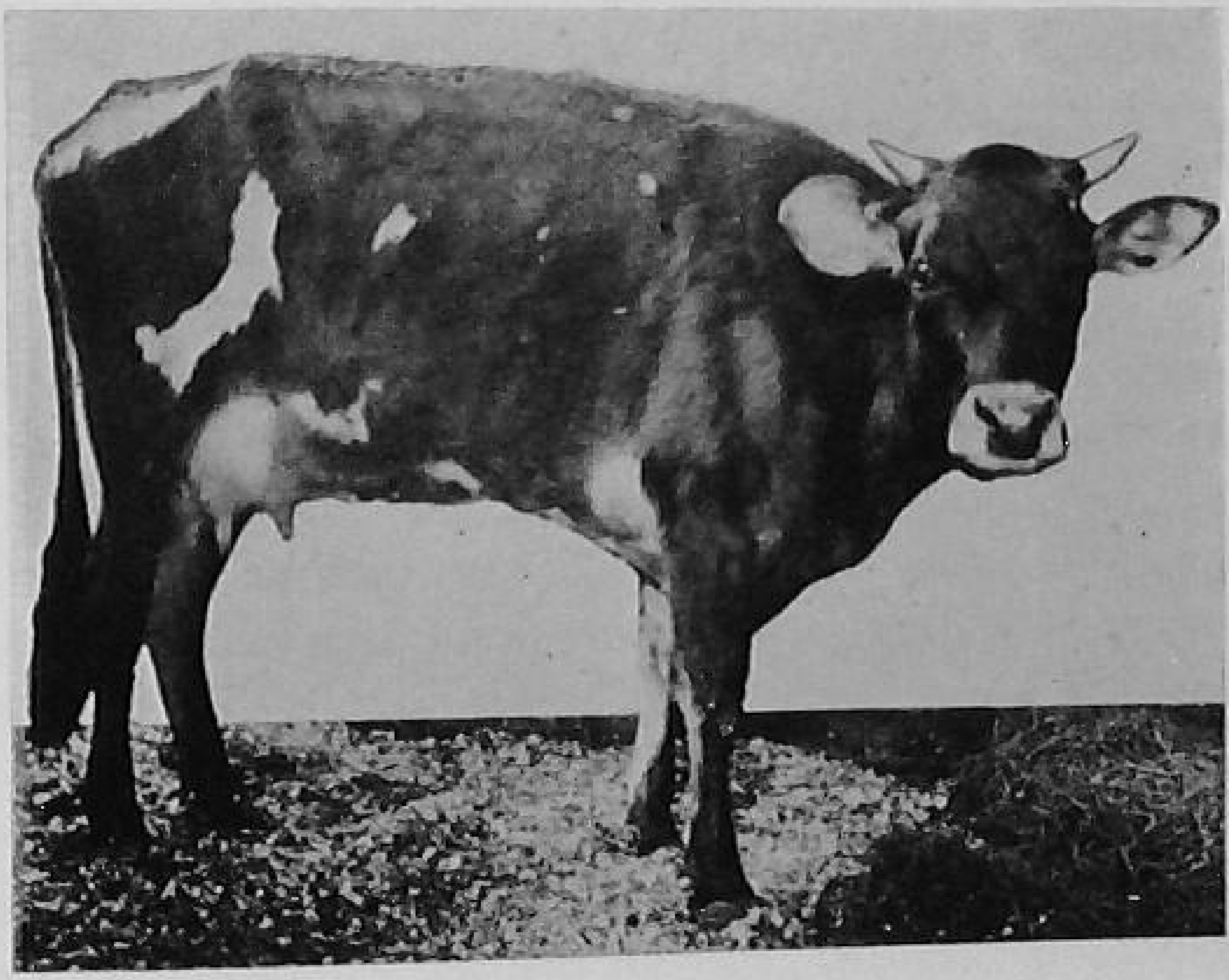

Fig. 62.—Acetonemia in two year old heifer, digestive type. (Photograph by courtesy of Dr. Jesse Sampson.)

A cow may be found down, unable to rise, and after a time regain her feet without help. In these cases of paresis there may be no apparent disturbance of the consciousness. A moderate degree of excitement and muscular twitching is occasionally present.

The course is from one to three or four days under appropriate treatment. Mild cases recover promptly. Emaciation and diminished milk flow may persist for several weeks. Recurrent attacks are not infrequent. Recovery is the rule, but a few fail to respond to treatment and become worthless because of emaciation and complete cessation of the milk flow. Occasionally a cow continues to milk heavily, even when emaciated from a prolonged attack.

The digestive type may be confused with traumatic gastritis, traumatic pericarditis, enteritis, or indigestion. Because of variations in

the course some authors have mentioned acute, subacute, and chronic types.

In the *nervous type* there is a marked decrease in the appetite, condition, and milk flow, combined with nervous symptoms. The attack is more severe than in the preceding form. Complete cessation of appetite and milk secretion is frequent. In a typical case excitation is marked; there are reckless, undirected, delirious movements, while the expression is wild and the eyes bulge. Various forms of motor irritation are present, such as sucking the tongue, rolling of the eyes, chewing movements, spasms of the muscles of the neck or back which cause an s-shaped lateral curvature, convulsions, walking in circles, treading with the feet, pressing forward in the stanchion, and champing of the jaws. Paresis is often present; this is manifested by staggering, leaning against the wall, legs straddled or crossed, drooling of saliva, and inability to rise. Paresthesia may be revealed by licking of the skin to the point of drawing blood, or the animal may lick the wall. Hypersensitiveness may cause the patient to bellow with pain when the skin is stroked or when the catheter is passed. Pinching the skin of the back into folds may cause the animal to go down. One or more of these nervous symptoms may be associated with either the digestive or milk-fever type, and the nervous type may occur in a mild form. Differentiation between the types is based on the nature of the predominant syndrome. While the extreme nervous type has been described chiefly as a parturient affection, receiving the name *mania puerperalis*, we have seen it more often as a nonparturient disease. In the past these cases have been confused with meningitis, cerebral hyperemia, and infectious meningoencephalitis in ruminants. In this type of hypoglycemia the blood glucose usually is lowered more than 50 per cent.

In the *milk-fever type* the symptoms of ketosis are in most respects identical with those of milk fever, and it is probable that both conditions exist together in the same patient. Ketosis is suspected when nervous symptoms, other than paralysis, are present. These are manifested by delirium, wild expression of the eyes, constant muscular twitching, plunging about when down and unable to rise, and hypersensitiveness. Failure to recover from paralysis after receiving milk-fever treatment, and recurrent attacks, suggest acetonemia. In our experience, the milk-fever type has not been observed in animals under four years old, while, of our total number of cases of acetonemia, 15 per cent have been in animals below this age.

The parturient milk-fever type of ketosis has been confused with septic metritis. When a case of milk fever did not respond to treatment and the cow was found to be affected with metritis, the paralysis

has been attributed to septic intoxication or infection; it is probable that some of these have been cases of acetonemia.

The symptoms shown in the digestive and nervous types are characteristic of ketosis; the symptoms in the milk-fever type cannot be differentiated from those of milk fever. Usually there are no distinctive symptoms of ketosis when it is associated with pneumonia, traumatic gastritis and other diseases; it is recognized only by an examination of the urine. The extent of the presence of acetone in the urine in such affections as pneumonia, traumatic pericarditis and gastritis, and other diseases has not yet been determined. It is fairly clear, however, that in certain cases of metritis and pneumonia, improvement follows

Interval between parturition and onset of disease.	Blood calcium mg. per 100 cc.	Blood sugar mg. per 100 cc.	Blood acetone mg. per 100 cc.	Urine acetone mg. per 100 cc.
No. 1, 6 days	9.5		43.96	
No. 2, 3 weeks	7.6	42.37	41.65	
No. 3, 5 weeks	9.3	31.45	63.85	1209.67
No. 4, 8 days	5.7	34.84	11.10	
No. 5, 3 weeks	3.32	85.84	30.80	267.85
No. 6, 24 hours	6.45	70.42	12.47	33.33
No. 7, 7 days	10.75	58.48	1.75	28.27
No. 8,	4.00	22.83	5.82	24.47
No. 9,	2.85	75.76	6.06	49.65
No. 10, 24 hours	3.80	55.56	10.00	51.70
No. 11, 13 days	5.22	71.43	4.48	4.48

Normal: blood calcium, 9-11; blood sugar, 40-60; blood total acetone, 2-6; urine total acetone, 3-15 (mg. per 100 cc.). Total acetone bodies are expressed as acetone.

the use of dextrose or other drugs which cause the acetone to disappear from the urine. For discussion on differential diagnosis, see milk fever.

The *urine* shows a marked increase of ketone bodies (acetones), amounting to as high as 1250 mg. per 100 cc., as compared with a normal average of 7 (Sampson-Hayden). When these bodies reach 15-20 mg. per 100 cc. of urine, one may conclude, according to Sampson and Hayden, that the condition is bordering on acetonuria. There is a similar increase in the *blood*, where the normal acetone bodies average approximately 3 mg. per 100 cc. of whole blood. When these total acetone bodies reach 10 or more mg. per 100 cc. of whole blood, one may conclude, according to Sampson and Hayden, that the condition is that of ketosis. The *milk* may be off flavor and react to the Ross test for acetone.

Analyses furnished by Sampson, Gonzaga, and Hayden[7] of the Department of Physiology on material from atypical cases of milk fever

suggest that blood changes characteristic of both milk fever and acetonemia may appear together in the same patient. This is illustrated in the table on page 383, based on 11 cases.

Numbers 1, 2, 3, 4, 5, 6, and 10 show an increase in the total acetone bodies in the blood. In addition, No. 5 shows hypocalcemia and hyperglycemia, which are characteristic of milk fever. Frequent variations in the results of treatment with calcium gluconate, inflation of the udder with air, and dextrose are possibly accounted for by variations in disturbance of metabolism.

Test for Acetone Bodies

Examination of the urine according to the following method, developed by Dr. Hayden, will indicate the presence of acetonuria. The reagents consist of (1) a mixture of one part of finely powdered sodium nitroprusside with one hundred parts pure ammonium sulfate and (2) a small supply of sodium hydroxide flakes.

One gram of the nitroprusside-sulfate mixture is dissolved in 5 cc. of the suspected urine, or milk, then a small flake (about one-fourth inch square) of the sodium hydroxide is added. A purple permanganate color indicates a positive test. Urine may be obtained either by catheterizing, or by slightly stroking the ends of the hair over the perineal region. The entire procedure requires not more than five minutes.

The color will grow more intense in the course of two or three minutes. The permanganate color is pronounced even in a moderate acetonemia but becomes more marked in severe attacks. A ketone test may be positive in any high-producing cow if she goes off feed or is not able to consume and digest enough food for her energy requirements, but high ketone values do not cause nervous reactions unless the sugar is low. A positive reaction may be considered as an indication of utilization of fairly large amounts of fat and a deficiency of carbohydrate available for metabolism. A positive Ross test is read one to four plus according to the density of the purple color. Roepke[13] writes that "the usefulness of the nitroprusside or Ross test for acetonemia may be greatly extended in cases where a positive test is obtained if it is repeated with a 1:10 dilution of the urine. Animals with a severe ketosis usually show a three or four plus reaction with a 1:10 dilution." Because of the high percentage of apparently normal cows found with ketones in the urine in herds under routine examination, doubt has been expressed concerning its value in diagnosis. One needs to consider, however, that in the absence of an associated syndrome the clinician does not encounter ketosis, and that the test is only a part of the evidence contributing to the diagnosis.

Treatment.—Prompt benefit results from the administration of dextrose.* Give 500 to 1000 cc. of a 40 per cent solution daily in the vein. One to five doses are required for complete recovery. One may also use a 20 per cent calcium gluconate solution. Give 250 cc. intravenously and 250 cc. subcutaneously each day. It may be necessary to repeat from one to four times.

Chloral hydrate, 30 grams once or twice daily is highly effective, especially in the nervous type; this may be given in a capsule, or, preferably, dissolved in one pint each of molasses and hot water. Chronic cases recover under the administration of 30 grams of chloral hydrate followed by 15 grams daily for six days.

Following the observation that propionic acid caused a marked elevation of blood sugar, Schultz[14] treated nineteen cows with ketosis by oral administration of sodium propionate at levels of ⅛ to ½ pound daily. All of the cows showed increases in milk production and improvement of appetite. At the end of ten days or less of treatment blood sugar values were normal in all cows. It is a frequent practice to administer a pint of 40 per cent solution of dextrose per vein, combined with ½ pound sodium propionate (Mycoban) daily for four days, either in the feed or by drench since it is very soluble in water.

On the theory that ketosis in dairy cows is due to an adrenal insufficiency involving the adrenal cortex and the anterior lobe of the pituitary gland, cortisone and ACTH have recently been used.[15, 16] The corresponding case reports are for the most part lacking for each individual, or show that other remedies were employed when response was unsatisfactory, or include too few cases for a disorder from which most individuals recover, or fail to mention complicating diseases. It is probable that these products will be included in the list of remedies of proven value in the treatment of ketosis.

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* A form of dextrose which is listed by some chemical supply houses as pure, anhydrous, granular dextrose (corn sugar) at 25 pounds for two dollars has been in use in our clinic for some time. It has been a satisfactory therapeutic preparation. The solution is made as follows: dextrose (C.P. anhydrous), 400 grams; distilled water to make 1000 cc. Add the dextrose to boiling water and continue to boil for 5 to 10 minutes after the dextrose has dissolved. Filter while hot into sterile bottles and add 4 drops of phenol to each bottle.

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TRANSPORT TETANY

(Railroad Disease; Railroad Sickness; Staggers)

Transport tetany is chiefly an affection of cows which presents symptoms similar to those of milk fever. The attack occurs during transportation or within twenty-four hours after arrival at destination. Susceptible individuals are pastured cows advanced in pregnancy and in good condition; it is less frequent in individuals that have recently calved. Thus it is a summer disease occurring chiefly from May to September. It has been reported in ponies following a railway trip, and according to Greig there is a disease in ewes apparently identical with transport tetany in the cow.

Etiology.—The disease is induced by transportation in hot cars or trucks without food or water. Especially predisposing are advanced pregnancy and an uninterrupted transportation period of twenty-four hours or more. In a series of 62 cases observed by Denker,[1] it was not seen in cows less than seven months pregnant; 18 developed in transit or within five to six hours thereafter. The attack is usually within twelve hours after unloading, but it may not occur until after forty-eight hours. As in milk fever, it attacks cows 6 or more years old and in excellent condition. The cause is unknown, but it is regarded as an acute hypocalcemia.

Morbid Anatomy.—There are no definite autopsy changes. In slaughtered cows Denker noted a brownish red to a dark-brown color

of the muscles, and a dark arterial blood which was especially marked in the coronary arteries.

Symptoms.—The early symptoms are anxious expression, restlessness, partial paralysis of the hind parts, and a stiffness of the tarsal joints. There may be clonic spasms, grinding of the teeth, and frothing around the lips. The nostrils are dilated and there is a marked increase in the rate of breathing. The cow staggers when attempting to walk, and finally she goes down and is unable to rise. The mucous membranes are congested and the pulse 100 to 120; fever is not the rule. There is a marked thirst but no appetite for food. Contractions of the rumen, and bowel evacuations are suspended; usually there is a retention of the urine. The rectum and bladder seem to be paralyzed. At first the consciousness is normal, but sopor rapidly develops and the cow may lie with the head around to one side, as in milk fever. The breath may have a distinct odor of acetone, being sweetish and similar to that of chloroform.

There may be marked improvement in four or five hours, or the condition may persist unchanged for three days, when slaughter is indicated. The prognosis is relatively favorable in cows that have calved, or that expel the fetus during an attack. Under other conditions it is doubtful or unfavorable. In general the prognosis is not good.

Treatment.—The introduction of calcium gluconate and other calcium preparations, as used in milk fever, has greatly reduced the mortality. Chloral hydrate 1 ounce (30 Gm.) may be useful in the stage of excitation.

Prophylaxis.—One or two days before transportation the animals should be removed from the pasture to a stable and given dry food. They should be loaded carefully and furnished hay in transit. Denker observed that the attacks were less frequent when cows were placed and kept in an open field immediately after unloading from the train.

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GRASS TETANY

(Lactation Tetany; Hypomagnesemia; Grass Staggers)

Definition.—Grass tetany is a highly fatal disease of cows and sheep occurring in cows principally in the spring during the first two weeks after animals are turned to lush pasture. It is characterized by tetany followed by convulsions and loss of consciousness. The blood serum is low in both calcium and magnesium. The mortality is high and death may result within an hour.

Etiology.—Grass tetany was first described by Sjollema[1] in Holland as a disease in cows that after recent calving were turned to lush pasture in the early weeks of the spring. An examination of the blood serum revealed that it was low in calcium and especially low in magnesium. The following reported values for blood from normal cows, those with milk fever, and with grass tetany were published by Sjollema and Seekles[2]:

	Normal	Milk fever	Grass tetany
Total calcium	9.35	4.35	6.65
Total magnesium	1.66	2.19	0.455
Total phosphorus	4.57	2.16	4.33

Whether the low magnesium value was the cause of grass tetany, the authors considered doubtful, as in tetany due to parathyroid deprivation in man, the magnesium content is normal, as is also the case in parathyroidectomized dogs. Perhaps the relation between the Ca and Mg is important.

The relation of Ca/Mg in the normal animals is $\frac{9.35}{1.66} = 5.6$

The relation of Ca/Mg in milk fever animals is $\frac{4.35}{2.19} = 2.0$

The relation of Ca/Mg in grass tetany animals is $\frac{6.65}{0.445} = 14.6$

The success of magnesium sulfate in the treatment of tetany is in agreement with these figures. It would appear, then, that the clinical picture of milk fever and grass tetany depends upon the relation between Ca and Mg and not on the value of these substances taken singly. They[2] consider that grass tetany is caused by a poison which is formed in the intestines.

In Scotland grass tetany was briefly described in 1931, by Lothian[3] as lactation tetany in pregnant and non-pregnant lactating cows on old pasture grazings in the winter and early spring months; the affected cows had been lying out or housed by night in cattle courts. The onset was sudden with intense tetany, retraction of the head, spasmodic twitching of the facial and other superficial muscles, with death in paroxysm in the course of a few hours. Because of the number found dead on arrival, anthrax was suspected. In less severe cases the symptoms were similar to those of milk fever. In one sample of blood the calcium was very low, and two cases treated with calcium gluconate recovered.

As reported in England by Allcroft and Green,[4] the disease was observed mostly in the spring months, but also in autumn rather than

spring, and in mid-winter amongst animals grazed all the year 'round, under climatic conditions which precluded "flush of grass" as a factor. In grass tetany there was a fall in both the Ca and Mg and in affected herds a fall in the serum Mg was present in a part of the apparently normal cows. "There was a regular seasonal variation in serum magnesium, with a maximum in summer to a minimum in winter and rising again through spring to a maximum again the following summer, without any clinical symptoms whatever. . . . The incidence of the hypomagnesemic clinical condition, associated either with normal calcium or with pronounced hypocalcemia in the 'mixed disorder,' varies widely from year to year. Thus, February 1932, was a bad season in Herefordshire leading to the loss of some 500 cows and to the addition of the term 'Hereford Disease' to the popular synonyms for the disorder."

In the United States it has been described by Udall[5] in Kentucky in a herd of 200 beef breeding cows on pasture during the winter months from December to April. They were in excellent condition because of supplementary feeding. Fifteen were affected and the mortality was about 50 per cent. As in milk fever, the clinical symptoms were usually associated with parturition, but a few appeared at any time during the lactation cycle. The disease was characterized by violent tetany followed by convulsions. The blood serum of the sick was low in both calcium and magnesium ("mixed hypocalcemia-hypomagnesemia"—Barker[6]). In the healthy cows the blood serum was low in magnesium and normal in calcium.

In the Great Central Plains, from the Mississippi to the Rocky Mountains, and especially in the South Plains, from late September to March thousands of cattle are shipped in to graze on wheat and the estimated losses from grass tetany ("wheat poisoning") in these areas are reported by Redmond[7] to exceed those from all other diseases combined. On wheat pastures grass tetany seldom develops sooner than after 30 days, and usually it is 60-90 days or longer. The most susceptible are either pregnant or in early lactation; open heifers and steers are seldom attacked before 120 days. Predisposing factors are debility and poor condition, as in cattle shipped from drouth areas, severe weather, involuntary exercise and excitement in changing pastures, and in transportation in cars or trucks. Depending on the distance, losses of 20 to 40 per cent in transit are not uncommon. It may also occur in cattle on oats, barley, rye, winter peas, and Bermuda grass.

Blood samples examined from many cattle that were shipped into the stockyards at Amarillo, Texas, and that came down with wheat poisoning gave the following results:

Mg. per 100 cc.	Normal	Sick
Sugar	80–120	40
Calcium	9–12	3.5–7
Magnesium	2–3	less than 1

"After cattle are placed on wheat the blood sugar progressively drops from a normal of 80-120 mg. per 100 cc. of blood until it levels off within 30 to 60 days at about 50 mg. per 100 cc. of blood. Symptoms of hypoglycemia develop if the blood sugar drops lower than 40 mg."

Such analyses suggested that the cause might be due to a deficiency of these elements (Ca and Mg), but the wheat plant contains more than enough of both elements to furnish the required amount, and additional amounts in the form of mineral mixtures failed to prevent the disease or affect the blood levels of calcium and magnesium. As expressed in the report by Sjollema,[2] it was thought that the disturbance in mineral metabolism was due to a magnesium-calcium ratio of 1:14 in place of the normal 1:3.5. There is some inhibitory influence upon the normal calcium and magnesium blood level, since adequate amounts of both are taken in by the animal. In the Texas report[7] it is suggested that an excessive intake of potassium (300 grams of potassium nitrate per day in a grown cow on wheat) may be responsible for the disturbed metabolism, and symptoms of grass tetany were produced by intravenous injections of large amounts of potassium chloride. While Redmond[7] (Texas) concludes that "the nervous excitability undoubtedly is due to the low magnesium level, it is our belief that continued excessive potassium intake in some way disturbs the normal mineral metabolism, thereby bringing on the condition known as wheat poisoning."

And Newsom[8] writes that, "As a consequence of all these observations tetany is associated with decreased calcium and magnesium in the blood serum but there is no assurance that it is due to this deficiency. Thousands of cases have occurred in cattle and sheep on the wheat farms . . . but as yet there is little accurate information on the cause."

Symptoms.—The symptoms of grass tetany are similar to those of milk fever combined with the nervous form of acetonemia. Of the three groups of nervous symptoms: deranged consciousness, motor irritation, and paralysis, the most striking in this affection are paresis and motor irritation. The symptoms vary widely according to the intensity of the attack. In a mild form one observes dullness, inappetence, and staggering or paresis. Within twenty-four hours distinctive symptoms appear in the form of trismus, grinding the teeth, twitching

of the muscles, anxious or wild expression, nystagmus, erect ears, tetanic contraction of the muscles of the tail and tetany of the hind limbs (tetanoid paresis), and frequent urination. The temperature is normal. From an early attack there may be inability to rise because of paralysis. Any disturbance may aggravate the symptoms of tetany and throw the animal into general spasms or convulsions. In Metzger's[9] case, for example, passing a urinary catheter caused severe general clonic-tonic contractions of the degree of convulsions. The cow rolled on her side, her legs threshed back and forth at a rapid rate, her head was drawn up and back as far as it could be drawn (orthotonus), the membrana nictitans protruded to completely cover the eyeballs, and the breathing dropped to two or three a minute. In *severe* attacks, paralysis and convulsions develop soon after the onset and excitement is intense. The victim may plunge around aimlessly, unable to rise (tetanoid paralysis), and often there is pounding of the heart.

Such attacks may result in death before discovery of the sick or in transit, or before the arrival of a veterinarian when anthrax may be suspected.[3]

Pulles[10] reports that in his practice the disease is almost enzootic in the spring after turning to pasture; that it occurs chiefly at from three to twelve weeks after calving; and that the symptoms in general are like those of milk fever, only more intense. The blood shows a slight decrease in calcium and a marked decrease in magnesium.

In the case described by Metzger there was a decrease in the serum magnesium, as well as in the calcium and phosphorus. The following table gives the blood analysis on the day of the attack, March 24, and on April 15.

	March 24	April 15
Cell volume	44%	30 %
Blood sugar	41 mg.%	45 mg.%
Inorganic P. on whole blood	2.3 mg.%	5.3 mg.%
Calcium	7.5 mg.%	9.6 mg.%
Non-protein nitrogen, whole blood	27.9 mg.%	26.0 mg.%
Serum magnesium	0.145 mg.%	2.5 mg.%
Test for acetone bodies	negative	

In the *chronic form,* high-producing cows show a gradual loss of condition, though the appetite and milk production may remain normal. Examination is negative and the condition may remain unchanged for weeks and months. Then the more typical symptoms appear, such as deranged motility, nervousness, and irregularity in the milk flow and appetite. Finally the cow passes into convulsions and dies, unless prompt treatment is available. Between the acute and chronic types there are transitional forms.

Treatment.—Since grass tetany is recognized as a form of tetany, it responds promptly to intravenous injection of calcium chloride. This is used in Holland as follows: calcium chloride (30 Gm.), magnesium chloride (8 Gm.), in 250 cc. water per vein. The salts are dissolved, filtered, and sterilized, and the injection is promptly beneficial for both milk fever and grass tetany. Pulles[10] reports the treatment of more than 100 cases, and Sjollema[1] writes that over 200 were successfully treated by this method in the spring of 1929. A second injection is rarely required. The magnesium prevents heart injury from the calcium. Injection is conducted over a period of ten to fifteen minutes, and the pulse is carefully followed in order to discontinue injection if the pulse increases to two or three times the normal. In such cases, which are unusual, the udder is inflated with air. The condition also responds promptly to subcutaneous injections of magnesium sulfate (200 cc. of a 25 per cent solution) repeated at short intervals as needed. Equally prompt results have been obtained from calcium gluconate.

Redmond[7] reports that calcium gluconate cures in a few minutes, and that somewhat better and more lasting results follow when the calcium gluconate is fortified with 8-10 Gm. magnesium sulfate. To avoid heart failure allow 15-30 minutes for the injection of 500 cc. Cases that have been down for any considerable period respond more slowly, and receive large quantities of saline intravenously, sometimes 5,00 to 8,000 cc. To restore appetite give ½ to 1 grain of apomorphine.

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TETANY IN CALVES

Hypocalcemia, as well as hypomagnesemia, has been observed in calves from three months of age and upwards when fed on an exclusive milk diet. In the beginning of the attack the affected animal walks stiffly, is nervous in its actions, and is easily startled when one approaches. Any sudden loud noise greatly aggravates the muscular twitching. In a 3-months-old calf there were symptoms of fright combined with spasmodic muscular contractions of the muscles of the neck and limbs. When by itself and not aware of being observed the calf would stand with mouth partly open or it would continuously open and close the jaws in an upward and downward direction with no horizontal movement. At the end of a week it fell in convulsions, bleating and stiffening when medicine was administered orally. It was standing again in fifteen minutes, and died that night. In such cases any unusual disturbance throws the animal into convulsions. A mate of this calf died in convulsions induced by the noise of coal passing through a chute. The blood calcium in one case was 7 mg. per 100 cc., as compared with a normal of 9 mg. The mortality is high, and no special lesions are found. This condition has been described by Duncan[1] et al., who report on consistent failure to raise calves to maturity on a ration of whole milk. This failure results from either hypocalcemia or hypomagnesemia, and it is practically impossible to distinguish between them unless blood studies are made. Calves which develop true blood calcium tetany invariably have blood calcium values below 7.5 mg. per 100 cc. Low magnesium tetany is infrequent, since it does not occur when the calf has access to roughage. The symptoms are irritability, nervousness, and anorexia. Apparently blind, the calf will run into obstacles or turn in circles. Finally there are convulsions with extension and contraction of the legs, and frothing at the mouth that may continue for several minutes. Young calves are able to withstand several such convulsions, but older calves usually succumb to the first attack. The lesions of the calves described by Duncan, et al.[1] have been described as follows by Moore, Hallman, and Sholl.[2] "The principal pathologic alterations consisted of a deposition of calcium salts in the yellow elastic fibers of the endocardium, of the aorta, jugular vein and large arteries, of the surfaces of the diaphragm, and of the trabeculae and capsule of the spleen. There were also present various degrees of hepatitis and nephritis." In calves observed in the ambulatory clinic the lesions have been those of white muscle disease and attributed to vitamin B_6 deficiency.

On a whole milk diet for growing calves, the addition of magnesium is called for, where during a long feeding period on an exclusively milk

diet the animals may get into a condition of tetany or convulsions—G. Bohstedt.[3]

The treatment is to keep the animals in a quiet place and administer chloral hydrate and vitamin E, as in white muscle disease.

Tetany and *convulsions* in newborn calves ascribed to recessive semilethal factors have been described by Richter.[4] From the time of birth such calves are subject to acute spasms. They are unable to rise, but the consciousness, reflexes, and appetite are normal. Convulsions may be induced by handling the calf. In one herd observed by the author, where recessive epithelial defects of the skin at the fetlocks and the mucous membrane of the mouth were present, four individuals presented the symptoms described by Richter.

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PREGNANCY DISEASE OF EWES

(Ketosis; Hypoglycemia; Hypomagnesemia)

Definition.—A disease of mature, well-nourished, pregnant ewes characterized by marked hypoglycemia, acetonemia, and acetonuria due to lack of carbohydrate. The chief symptoms are twitching of the ears and body muscles (motor irritation), paresis, depressed consciousness leading to coma, and rapid emaciation. The mortality is 90 per cent or more, and extreme fatty infiltration of the liver is found on autopsy.

Etiology.—Pregnancy disease is a serious problem wherever sheep are raised. In North Dakota losses from 1 to 25 per cent of a flock are common—Roderick,[1] and it stands third as a cause of death in the autopsy records of the Michigan State College—Sholl. It occurs in stabled farm flocks rather than on the open range, and chiefly in twin pregnancy. While opinions on the cause are somewhat contradictory, there is general agreement on the conclusions of Groenewald et al.[2] of South Africa that the disease is a carbohydrate depletion induced by the requirements of pregnancy, similar to the carbohydrate depletion of lactation in acetonemia in cows. They dispose of the idea

that it is caused by toxins, since tissue and blood changes identical with those of pregnancy disease have been produced experimentally in nonpregnant ewes; they are skeptical in regard to the influence of lack of exercise which may be confused with reduced intake due to change of food or inclement weather; but either inadequate ration or semi-starvation in fat pregnant ewes is regarded as a potent factor. In experimental fat pregnant ewes symptoms of pregnancy disease appeared within three days after a change to poor hay only.

The current view as expressed by Roderick is that twin pregnancy imposes metabolic requirements beyond the capacity of a ewe on insufficient diet. This lack of capacity is observed especially in the liver which has been depleted of its glycogen to maintain the blood-sugar level of the body. In the report by Dimock, Healy, and Hull[3] little emphasis is placed on lack of exercise as a cause. "In those flocks in which acidosis or pregnancy disease developed, it was found that the ewes were on winter pasture, possibly being fed a little corn stover or a small amount of corn. In a few instances the ewes received corn and oats. In very few flocks where the disease developed, the ewes received what was apparently a proper balanced diet."

Leslie,[4] in New Zealand, also expresses the conviction that apart from big lambs *in utero*, the chief factors are (1) underfeeding and (2) unbalanced feeding. He believes that exercise has nothing to do with the prevention of the disease, and states that he has never diagnosed a case that was due to excessive fatness.

Because of certain exceptions in the blood analyses, the authors conclude that "These anomalous examples show that there is no direct correlation between symptoms on the one hand and acetonemia and hypoglycemia on the other hand and we are still in doubt as to the actual cause of the symptoms."[2]

Morbid Anatomy.—The gross lesions are confined chiefly to the liver which is yellow and friable. There is an extreme fatty infiltration. On the significance of the fatty liver, Clark[2] quotes from M'Fadyean that "towards the end of pregnancy, even in normal conditions, there is a tendency towards fatty infiltration of the liver that approaches the pathological," and comments that "whether the fat actually represents a morbid condition or not, therefore, becomes a matter of opinion as to the amount. . . . The fact that there is no visible damage by the fat and no icterus makes one hesitate to look upon this lesion as purely pathological. Yet when the liver has the color, feel, and consistency of butter, it cannot be denied that this condition has passed well over the border of pathological." Other gross postmortem changes are "fat necrosis, especially of the perirenal and omental fat, fatty

changes of the renal cortex and adrenal cortex, and atrophy of the lymph glands." Sections of the kidney show the presence of fat droplets in the epithelial cells of Henle's loops, a nephrosis—Roderick;[5] but these changes are a result and not a cause of the disease. Nonpregnant ewes on a starvation diet may develop changes in the tissues and in the blood identical with those of pregnancy disease, but the clinical symptoms are lacking. Clark[2] concludes, therefore, that "there is no known pathognomonic lesion, or group of lesions, by which a definite diagnosis can be made. . . . The only criterion we can go on is the presence or absence of symptoms before death."

Symptoms.—At the onset there may be twitching of the ears, muscular spasms, and a nervous expression. There is complete lack of appetite, the temperature remains normal and there is no jaundice. In a flock of sheep, where the initial symptoms may be unobserved, the onset appears to be gradual in the form of dullness, the affected sheep remaining outside the general flock. As the disease progresses there are symptoms of deranged consciousness and motor irritation, such as walking in a circle or standing with the head pressed against some object. Thus the symptoms and seasonal occurrence are almost identical with those of circling disease in sheep. The head may be drawn backward or to one side. Later the animal goes down usually lying with the head turned to one side, and is unable to rise without assistance. Coma finally develops, and disturbance in this stage may result in spasms and convulsions. Other symptoms are increased thirst, grinding of the teeth, and blindness. Labored and extremely rapid breathing may be present to compensate for the acidosis. Urination is frequent and bowel evacuations are somewhat suppressed. Expulsion of the fetus is followed by rapid recovery, if the disease is not already in an advanced stage. Because of marked increase in acetone in the urine in the early stages of the disease, the acetone test is a reliable means of making a diagnosis. Any hope for treatment depends on early diagnosis. To identify such cases, Leslie[4] submits ewes in affected flocks to forced exercise, which brings out the prodromal symptoms of impaired gait and motor irritation. Once the disease is advanced the case is hopeless. The course is from one to six days.

The blood shows a marked increase in acetone (acetonemia), and a marked decrease in blood sugar (hypoglycemia). The urine of normal ewes contains not more than a trace of acetone, while the urine of pregnancy disease contains from 10 to 300 mg. per 100 cc., or as much as 300 times more than the normal—Roderick.[5]

Treatment.—Leslie[4] reports recovery in about 40 per cent of the

cases treated. He administers molasses ("treacle") (1½ pounds for big ewes, ¾ to 1 pound for medium ewes, and ½ to ¾ for small and weak ewes); in addition they may receive a hypodermic injection of hypotonic solution of glucose (℥ xvi). While treatment is said to be useless, unreported observations by Sampson and Hayden[6] suggest that daily intravenous injections of dextrose (gm. 25 in 500 cc. of water) is beneficial if given early. Clark[7,8] of South Africa recommends, in addition to glucose, either lentin (1 mg.) subcutaneously or linseed oil (120 cc.) for relief of the ruminal atony and constipation of the large intestine, which is an almost constant finding in pregnancy disease and no permanent improvement can be expected until this is relieved; he also advises the intramuscular injection of vitamin B_1 (thiamin) daily. Insulin has also been used in combination with glucose.

Prevention.—Prevention is accomplished by providing an adequate diet during the last two months of pregnancy. Most cases of pregnancy disease occur in ewes in poor condition on a diet that is insufficient either in quality or amount. A lesser number occur in fat ewes where the disease results from interruption of an adequate diet; this may be due to storms or weather conditions, or any change in feed that interrupts the normal routine. Once a sheep stops eating from any cause it may continue on a restricted diet even when food is available. Drastic reduction in the diet of a fat ewe may induce pregnancy disease in less than a week. When available, nothing is better than good pasture.

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RICKETS, OSTEOMALACIA, OSTEOFIBROSIS

(Aphosphorosis; Acalcicosis; Ostitis Fibrosa)

Definition.—Deficiency diseases of the osseous system of ruminants are caused by a lack of either phosphorus or calcium. These deficiencies cause two different pathological conditions that may result in similar or identical symptoms and consequently lead to confusion over the diagnosis. *Phosphorus deficiency* in growing cattle causes *rickets*, a disease in which there is an excess of newly formed bone that fails to calcify (osteoid tissue). Phosphorus deficiency in mature animals causes *osteomalacia*, a condition in which there is a resorption of the bone minerals and replacement of normal bone with osteoid tissue ("adult rickets"). In both diseases there is a deficiency of inorganic phosphorus in the blood serum. *Calcium deficiency* causes atrophy and softening of the bone (*osteoporosis*). As a deficiency disease it is relatively infrequent in ruminants in this country; it is a part of the pathology of fluorosis.

While a great excess of either calcium or phosphorus interferes with the absorption of the other, wide differences of opinion have been expressed concerning the influence of such imbalance. Extensive observations by Theiler and associates[1], however, have led them to believe that in ruminants the amount of the deficient element and not the proportion is of chief importance. Theiler produced the disease (rickets, osteomalacia) both with a ration deficient in CaO and P_2O_5 and with a ration rich in CaO and poor in P_2O_5, and the lesions were in both instances, of the same intensity. He was unable to produce the lesions with a ration which was poor in CaO and rich in P_2O_5. The quantity of CaO was as low as 8.4 gr., and he doubted whether such a low calcium figure would occur under natural conditions. Rickets and osteomalacia under South African conditions is a pure aphosphorosis.

Since the terms rickets and osteomalacia are often applied in a broad sense to any symptom of mineral deficiency, as well as to diseases of the bones and joints, additional confusion is added to a subject that alone is complex. In all of these conditions there may be perverted appetite (pica), atrophy and softening of the bone, stiff gait, and a tendency to fracture. An accurate diagnosis may depend on a chemical examination of the blood, or a microscopic examination of the bone.

Etiology.—*Phosphorus deficiency (aphosphorosis)* occurs in animals on a diet composed chiefly of roughage, such as hay, straw, or pasture. Other common roughages low in phosphorus are corn silage, beet pulp, roots, and molasses. Hay alone contains sufficient phosphorus when grown upon normal soil, but on phosphorus deficient soil may contain not more than 0.142 per cent P_2O_5 as compared with a normal of

0.44 per cent on unaffected areas—Eckles.[2] Roughly speaking we expect aphosphorosis in a mild form (osteophagia) when the P_2O_5 expressed on the dry matter of the winter grass falls below 0.2 per cent, and in the acute form (rickets and osteomalacia) when the phosphorus drops to about 0.1 per cent or less of the dry matter of the mature grass (Theiler).[1] The grains are relatively high in phosphorus and their concentrates are still higher. The daily phosphorus requirement for a normal growing bovine is estimated at about 10 grams.

The P/Ca equivalent of the different foods is as follows: ordinary hay, 0.82; green grass, 0.63; green alfalfa, 0.21; wheat, 8.6; rye, 8.7; barley, 7.1; oats, 7.4; potatoes, 5.8; sugar beets, 1.1; linseed meal, 2.10; cottonseed meal, 9.95; and peanut meal, 30.78 (Mollgaard[3]). Thus hay and alfalfa are rich in calcium, when grown upon properly fertilized soils, while the grains are rich in phosphorus.

Aphosphorosis occurs chiefly in certain areas where there is a deficiency of phosphorus in the soil. In the United States such areas are found in Minnesota (Eckles[2]) and Texas (Schmidt[4]) and the largest known area is in South Africa. In the United States it has been reported by Mitchell and McClure[5] that aphosphorosis has been definitely recognized in Montana, Minnesota, Wisconsin, Michigan, Kansas, Utah, California, Texas, and Florida, and perhaps less certainly in New York, Pennsylvania, West Virginia, South Carolina, Alabama, and Mississippi. A report by Forbes and Johnson[6] upon phosphorus deficiency among cattle in Pennsylvania may be accepted as descriptive of the condition in the northeastern part of the United States. They state that in Pennsylvania there is no known area of low phosphorus content of the soil, yet "phosphorus starvation may be encountered anywhere, as a result of improper feeding, regardless of the composition of the soil and of the locally grown feeding stuffs," and that "definite phosphorus deficiency is a rare disease in Pennsylvania." There may be phosphorus deficiency on certain farms where no mineral fertilizer has been used for years, and on some of these farms in New York State where cattle have been found with symptoms of osteomalacia, the roughage has been found to be extremely low in phosphorus.

In investigations on osteodystrophic diseases in cattle conducted by Theiler[20] and associates, they observed that "rickets or osteomalacia is invariably brought about by diets low in phosphorus, the severity of the condition depending on the degree of the deficiency, the phosphorus requirement of the animal, and the duration of the experiment, while parallel experiments on group mates of the animals used for the phosphorus deficiency work, have not in a single instance produced rickets or osteomalacia when the diet was low in calcium and contained

sufficient phosphorus. . . . It is believed that the effects of phosphorus deficiency under practical conditions are aggravated by relatively high calcium which is almost invariably present in phosphorus deficient pasture."

Calcium deficiency (acalcicosis) may occur when the roughage is poor in quality, when grown on soil deficient in calcium, or when too little is fed. Bone deficiency develops less rapidly on low calcium diets than upon those low in phosphorus; this is due in part to the less complex and varied function of calcium in the general metabolism.

According to Sjollema[7] and others these diseases in herbivorous animals and swine are also caused by diets in which the Ca:P ratio is defective, as 1:9 instead of 1:1-2, and by lack of vitamin D. It is probable that the etiology varies somewhat in different parts of the world, according to different kinds of food, qualities of soil and water, and methods of fertilization of the soil. Sjollema[7] states that the frequency of rickets and osteomalacia seems to have increased with the intensification of agricultural production. He attributes this condition in Holland to changes in the type of winter rations used in high milk yields. Most farmers give only a little hay, together with concentrates such as extracted soy bean meal, peanut cake, etc., and large quantities of maize and other cereals. The percentage of calcium is low and that of phosphorus somewhat high; the Ca:P ratio may be 1:6 or 7, or even greater, instead of about 1:1. Diets in which the Ca:P ratio is the same as in some modern winter rations are often used to produce experimental rickets. In a report on similar feeding in Denmark, Mollgaard[3] writes that experience has shown the need of 60 grams calcium carbonate daily for cows that receive roots and only 4 to 6 pounds of hay daily. If the roots are replaced with grain, then give 60 to 70 gm. of calcium carbonate. Cows have received as high as 200 gm. CaO daily with no ill effects upon the milk flow. The detrimental effect of heavy feeding of concentrates without the proper quality of roughage has been described by Reed and Huffman.[8]

Observations on the production of experimental rickets in calves from which roughage was withheld have been described by Bechdel and associates[9] and by Gullickson and coworkers.[10] In these experiments the calves were fed little or no roughage and the diet was not physically suitable. Three calves described in Pennsylvania Bulletin 291 were fed a rachitogenic diet of concentrates and skimmilk. They were protected against rickets by a supplementary diet of oat straw of which one ate only 9 pounds before it was 100 days old. These examples show the extreme deprivation necessary for experimental production of symptoms of rickets in calves by withholding the ordinary sources

of calcium. That a supplement of vitamin D may correct mineral metabolism under such deprivation fails to establish the need for its addition to the diet under less extreme circumstances. The composition of "rachitic" diets used in some experiments is not unlike that of starvation diets. Rickets in calves under farm conditions where a winter ration was high in grain and low in roughage has been described by Hibbs et al.;[11] "The serum phosphorus and calcium were low and the serum phosphatase was extremely high—typical of rickets. Feeding of irradiated yeast brought about an improved condition."

It is probable that the deficiency disease in cattle on a low calcium diet, as described by Sjollema[7] and others, is osteofibrosis and not rickets, and that it is caused by a calcium deficiency and not by an imbalance of the Ca:P ratio, or a lack of vitamin D.

Experimental osteoporosis and osteodystrophia in calves and heifers on a calcium deficiency diet of 6 Gm. CaO and 25 Gm. P_2O_5; and of 3 Gm. CaO and 30 Gm. P_2O_5 have been reported by Theiler et al.[20]

Areas of calcium deficiency in the soil are much less than those of phosphorus. Calcium deficiency disease in cattle on the Coastal Plains of Florida has been described by Becker,[12] and it is probable that certain farms or other unidentified areas are also deficient or that deficiency may occur when the roughage itself is normally low. A useful table of the approximate percentages of calcium and phosphorus in different feeds has been published by Morrison[13] in Feeds and Feeding.

The belief is rather general that an unthrifty condition in calves and young stock housed in dark stables in the winter is an obscure subclinical form of rickets. Maynard[14] writes that rickets is very common in calves, and in an article by Miller[15] of the Royal Veterinary College in London one reads that "calves which have to spend the first four to six months of their life mainly indoors with a minimum of milk are not generally reared to best advantage. Definite clinical rachitis in either foals or calves, though by no means unknown, is not common, but I am satisfied that a condition akin to this disease, what I would like to call subclinical rachitis, occurs in a large proportion of winter reared calves. Its manifestations are general dullness and lack of normal growth and development, abdominal distension ('pot belly') stiffness on rising, lack of bloom, inferior development of the triceps, gluteal and gastrocnemius muscles."

In the diagnosis of rickets in children advantage is taken of the fact that the concentration in the blood serum of either calcium or phosphorus or both is always below normal while the rachitic process is advancing. This deficit in the serum is regarded as the chief cause of

the skeletal lesions. The product of the calcium and phosphorus expressed in milligrams per 100 cc. is accepted as an index to the presence or absence of rickets. In most cases the inorganic phosphorus alone is low. I have not found records of the phosphorus-calcium content of blood serum in this so-called subclinical group of rickets in calves, but it has been reported by Theiler,[1] that "Inorganic phosphorus of the blood of animals on a phosphorus deficient diet dropped markedly long before clinical symptoms appeared; this is a guide to the phosphorus deficiency of the pastures and an indication of the aphosphorosis in the animals themselves."

Concerning the influence of vitamins, Theiler[1] agrees with Stang (Berlin) that their importance in relation to mineral metabolism is somewhat exaggerated.

Osteomalacia in horses has been described by Kintner and Holt[16] in an extensive report on the affection in army horses in the Philippines. Their observations support the view that in this species the cause is a wide calcium-phosphorus ratio. There was a striking reduction in the cases following a change of ration when finely ground limestone was added to the food and the calcium-phosphorus intake ratio was reduced to approximately 1:1. In the different army posts the food rations were identical, but at Fort Stotsenburg, where the records showed the highest incidence of osteomalacia, the water contained the lowest calcium content (9.7 parts per million). At Fort Mills, where osteomalacia did not occur, the drinking water showed the highest calcium content of any post in the Philippines (125 parts per million). No evidence was obtained that equine osteomalacia is due to disturbances of internal secretions, parasitic infestations, heredity, infections, or vitamin deficiency. Kintner and Holt[16] conclude that "the production of equine osteomalacia was due to a ration containing a large amount of phosphorus in proportion to the calcium intake of the animal. It has been shown that the amount of calcium intake is of secondary importance to the calcium-phosphorus ratio. Osteomalacia will develop in horses when the ratio of calcium oxide to phosphorus pentoxide is 1:2.9. This condition did not develop during the nine-month period when the ratio was 1:1.9. . . . Feeding experiments with affected animals showed that the condition was arrested and anabolic processes stimulated when the ratio of calcium oxide-phosphorus pentoxide intake was 1:1.4. The condition progressed in affected animals when the ratio was 1:2.3."

According to Theiler[1] (1931) the abnormal skeletal condition in equines commonly termed osteomalacia is "osteodystrophia fibrosa," (Osteitis fibrosa). In South Africa the disease occurred sporadically in

stables but never in grazing horses. In goats the lesions are similar to those in equines.

The relation of osteomalacia to skeletal diseases of horses and mules, as represented by the spavin group of lamenesses, and described by Williams[17] and coworkers, and Greenlee,[18] remains to be established. It is generally conceded, however, that spavins and ringbones are a manifestation of a general disorder of metabolism affecting the entire skeleton.

From these somewhat conflicting opinions one seems justified in the conclusion that rickets and osteomalacia in cattle and swine are usually due to an absolute rather than a relative deficiency of minerals, and that while vitamin D is an essential factor it is not a deficiency factor in these animals. Observations made upon one species are subject to error when transposed to individuals of an entirely different species.

In the Philippines it has been observed that mules and native animals are more resistant than imported horses to osteomalacia.

Morbid Anatomy.—Since rickets, osteomalacia, and osteofibrosis are fatal only when marked changes in the skeleton have developed, an autopsy is rarely required to establish a diagnosis. Postmortem examination of advanced forms reveals distinct deformity of the long bones. On section of the bones the marrow is red and sprinkled with hemorrhages, while the cortex is thin, spongy and soft, so that it can be easily cut with a knife. The flat bones are fragile and easily bent. In equines and swine there may be a symmetrical enlargement of both sides of the face; the periosteum is easily removed and the underlying surface of the bone is red, while the external bony plate is easily cut. In the description by Kintner and Holt,[16] emphasis is placed upon variations in degree of change in parts of the skeleton subject to mechanical irritation and strain. Thus a deformity of the lower jaw was noted in practically all cases, there being a thinning and softening of the cortex together with a honeycomb appearance of the medullary portion. In many cases the vertebrae showed extreme thinning of the cortex and intervertebral discs. In all cases autopsied, severe joint lesions were noted in the form of erosions on the articular surfaces, and thickening of the synovial membrane.

To distinguish between rickets and osteofibrosis it is usually necessary to make a histological examination of the diseased bone.

Symptoms.—In general, chief emphasis has been placed upon the obvious skeletal changes as a measure of the injury caused by this disease. In recent reports, however, a number of investigators have expressed the opinion that the chief damage is not in the skeletal system, but in a lowered resistance. According to this view an obscure

form of the disease is relatively common and generally unrecognized. Nearly all writers speak of its increasing frequency, and there is a possibility that more regard should be given to mineral deficiency whenever there is evidence of chronic malnutrition. There are numerous reports, on the other hand, that improved cultivation of the soil has led to the disappearance of osteomalacia in areas where it was formerly prevalent. According to Maynard (personal conversation) a decrease of phosphorus in the blood serum from 6 mg. per 100 cc. to 3 mg. per 100 cc. in calves is an indication of rickets. Such animals are unthrifty, and they may develop large joints and show a depression of the back in the dorsal region.

In the cattle observed by Eckles[2] in Minnesota, there were crooked legs and in some a cracking sound in the joints. Animals grown in deficiency areas were decidedly stunted in size, and when mature the heads appeared to be too large for the body. Four-year-olds weighed less than 600 pounds. Cattle often were listless, even when there were no signs of deficiency. Losses were due to lack of growth, production, and reproduction. Stiffness of the limbs, swollen joints, fractures, perverted appetite, bone chewing, and abortion were common. Cows in milk suffered most severely and young growing animals came next. Convulsions were infrequent. In some, the tail became so soft that it could be coiled like a rope. When the depraved appetite was marked, sterility and abortion were most frequent. Cows brought to the deficiency areas developed the symptoms in about a year. Other symptoms were recumbency, arched back, groaning during defecation and urination, and pain on pressure over the back. Cattle were most subject to the disease in the late winter and spring; they were most severely affected when the diet was largely native or wild hay, though cases were observed when alfalfa was fed in abundance. In sheep wool-eating is a prominent symptom of mineral deficiency. Osteophagia (bone-eating) is an indication of phosphorus deficiency, but it may be absent when a deficiency exists, and it may be present in a variety of other conditions.

In cows fed upon a phosphorus deficiency ration consisting of poor roughages, such as cereal straw and corn stover, the symptoms are poor condition, failure of estrum, atony of the digestive system, low milk production, weakness and symptoms of starvation. Occasionally there are symptoms of pica, as shown by chewing wood, bone and other materials, as well as stiffness, and even fracture of the femur.

In the Philippine cases there was a serum calcium deficiency of approximately 9 per cent, while the inorganic phosphorus of the serum showed an increase of 20 per cent (Kintner and Holt).[16] Individual

resistance was extremely variable. Some animals developed the condition within six months after arrival in the Philippines, while others maintained under similar conditions for more than five years did not develop the disease. The earliest symptoms were changes in activity, and the development was gradual in the form of intermittent and shifting lameness. As the disease progressed the lameness became more marked. Perverted appetite was not noted as an early symptom. Bony enlargements of the mandible were present in 98 per cent; it was best detected on the lateral border adjacent to the molar teeth. The changes of the blood serum varied; on some days the elements were present in the blood in normal amounts. Spavins and ringbones were frequently present.

Treatment.—In the prevention and treatment of osteomalacia and rickets in herbivora and swine an adequate supply of the deficient mineral has proved to be effective. This may be furnished in the diet, or in special preparations containing calcium or phosphorus. Since the vast majority of clinical cases of mineral deficiency that have been reported in herbivora have been caused by lack of phosphorus, satisfactory results have followed the selection of food rich in phosphorus, or the administration of bonemeal. Foods rich in phosphorus are the cereals, such as wheat, barley, oats, and cottonseed meal. Peanut meal is especially high. In South Africa the condition was overcome by Theiler by feeding wheat bran, bonemeal, sodium phosphate, and even phosphoric acid. Theiler[19] (1924) states that "any digestible phosphorus compound, given as a supplement to the natural grazing, rectifies the deficiency and permits of normal development. . . . Bonemeal feeding is the practical solution for ranching conditions. Bran is useful for dairy stock or high-grade animals receiving supplementary rations in the ordinary course of events. Rock phosphate is of little value and presents difficulties in administration." In Africa, the bonemeal requirement varies from ½ pound per head per week for adult oxen and young calves, ¾ pound for growing stock over 300 pounds in weight, up to 2 pounds or even more for lactating cows. It is recommended that this be given in daily portions, or at least tri-weekly. For milking cows, Forbes regards steamed bonemeal (2 to 6 ounces per day) as the most effective mineral supplement.

Eckles[2] failed to obtain benefit from calcium carbonate or cod liver oil. Where cows had free access to steamed bonemeal in mineral deficiency areas, the average consumption during the winter was 3.57 pounds each per month, and all symptoms of osteomalacia disappeared. Experimental animals with a history of depraved appetite developed a depraved appetite on a basal ration composed of roughage and oats

from deficiency areas; these symptoms also appeared when the basal ration was supplemented by the addition of calcium carbonate and cod liver oil. Symptoms failed to appear when the ration contained mono-basic sodium phosphate (100 grains daily). Recovery from depraved appetite was rapid when affected cows received either tricalcium phosphate or mono-basic sodium phosphate; they also gained in either body weight or milk or both.

In the Philippines, where osteomalacia in horses is due to a deficiency in calcium, Kintner and Holt[16] reported marked improvement after addition to the ration of finely ground limestone (36 grams daily). They believed that improvement depended on a ration in which the calcium-phosphate ratio was 1:1. When this ratio was 1:25 the disease progressed, even when the absolute calcium requirement was sufficient. Thus improvement did not occur when calcium phosphate (100 grams daily) was substituted for finely ground limestone (calcium carbonate). Opinions differ, however, over the need of a definite calcium-phosphorus ratio when the absolute amount of calcium and phosphorus is sufficient.

Because of the enormous consumption of calcium by lactating cows, it is generally assumed that this material should be added to ordinary rations. Thus Mollgaard[3] writes that in Denmark experience shows the need of 60 to 70 grams daily of calcium carbonate for cows receiving grain and 4 to 6 pounds of hay daily. It seems improbable that mineral deficiency may be a contributory cause of sterility, retained placentae, and other reproductive disturbances so common among cows fed to capacity for high milk production. Yet the mineral requirements of such cows may be higher than is commonly estimated. To meet these requirements, 1 per cent bonemeal is usually included in commercial dairy feeds. We do not have complete knowledge of the mineral requirements of domestic animals.

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RICKETS, OSTEOMALACIA AND OSTEOPOROSIS IN SWINE

(Posterior Paralysis)

Definition.—The definitions of mineral deficiency in swine are the same as for bovines; a deficiency of phosphorus causes rickets, while a deficiency of calcium causes osteoporosis. As in other species, the term rickets is commonly applied in a broad sense to any disease in swine that presents the clinical syndrome of rickets.

Etiology.—In experiments conducted in South Africa, osteodystrophic diseases were produced only when the intake of phosphorus or calcium or both was low. In 1937 Theiler et al.[1] reported the following results of feeding experiments in which various combinations of calcium and phosphorus were fed to pigs: 1. Low phosphorus with abnormal ratio caused severe rickets with low serum phosphorus; light was without effect. 2. Low calcium with abnormal ratio caused osteoporosis with bone atrophy but no rickets, serum calcium was normal;

light was without effect. 3. Low calcium and phosphorus with normal ratio caused marked bone atrophy and a suggestion of rickets; the detrimental effect was less severe than that of a deficiency of either calcium or phosphorus with an abnormal ratio; absence of light was detrimental. 4. Sufficient calcium and phosphorus with abnormal ratio, no change.

Mineral deficiency diseases are far more frequent in swine than in other domestic animals. This is explained by their limited variety of diet on many farms, and especially on small farms where pigs are often fed whatever is least expensive, as swill, skim milk, corn, wheat midlings, etc. It is estimated by Mitchell and McClure[2] that growing swine require 0.3 per cent of calcium and 0.35 of phosphorus expressed as a percentage of the ration fed.

The more important facts concerning swine rickets have been demonstrated by Forbes.[3] In 1914 he reported that in growing pigs on practical rations all of the seed products were inadequate sources of calcium. The pigs stored from nine to ten times as much calcium from rations containing milk and tankage as from the best ration composed of grain alone. He interpreted these results as emphasizing the importance of pasture, forage crops, and dry roughage, especially legumes. In 1915 he showed that the combination of protein with corn and insufficient minerals, also resulted in rickets.

In 1922 McCollum et al.[4] reported that the addition of precipitated calcium carbonate or of pulverized limestone to cereal rations caused marked increase in retention of calcium, magnesium, and phosphorus. The addition of lime to grain rations increased the density and strength of the bone, but did not influence the growth. Among the different preparations used, steamed bone produced a maximum of hardness, while rock phosphate produced weak bones.

Considerable has been written on the deficiency of vitamin D (the "rachitic factor") as a cause of rickets in swine. Such deficiency is alleged to occur when swine are deprived of sunlight in dark pens in the winter months, if the food is lacking in vitamin D. It is now generally believed that the essential cause of rickets in swine is a deficiency of calcium or phosphorus, chiefly the former, and that when sufficient minerals are provided, rickets and osteomalacia will not appear, even in the absence of vitamin D. If sufficient minerals are not available, these diseases may develop, even in the presence of vitamin D. In experimental feeding projects conducted at the Nebraska Experiment Station,[5] pigs upon a low calcium diet were protected against rickets by adding 1 per cent cod liver oil to the ration, and similar results have been observed by others. Regardless of these experimental observa-

tions, vitamin D should be considered as a possible influence only when there is a deficiency of calcium in the diet.

In South Africa, in areas where the dry season lasts for six or seven months, and little or no grain is available, avitaminosis A has been described by Kellerman[6] and associates. In experimental pigs the symptoms were similar to those of rickets: inappetence, lack of thrift, dry scaly skin, conjunctivitis, fits, posterior paralysis, and eating dirt. A posterior paralysis in hogs due to lack of vitamin A has also been reported by Hart and Guilbert.[7]

Morbid Anatomy.—The apparent lesions are confined to the joints and bones, the most marked changes being found in the epiphyses of the long bones, and in the ribs. Changes are also commonly found in the articular cartilages. The bone tissue may be so soft that it is readily cut with a saw or knife. In the marrow of the epiphyses of the long bones one often finds foci of hemorrhage. Healed fractures of the ribs, as well as periosteal and subperiosteal hypertrophies, are not infrequent.

The articular changes are variable in distribution and extent. The favorite joints are the shoulder, elbow, carpus, hip, stifle, and tarsus. Often there are no obvious changes in other parts of the skeleton. Within the joints there may be wrinkling and furrowing of the articular cartilage, and roughness or erosion of the articular surface. The joint capsule may be thickened and present villous growths on the synovial surface. There is no increase in the amount of synovial fluid. The ends of diseased bones are easily penetrated with a scalpel. When a rib is bent at right angles to the vertebral column it will bend and break like a piece of cardboard, while a normal rib will crack and splinter (Kernkamp).[8] In extreme cases, other skeletal structures, especially the head, have undergone marked changes, and the tarsal or carpal joints may be rigidly flexed. Fractures of the long bones and vertebrae may occur.

Symptoms.—The symptoms are chiefly a loss of function of the limbs; this may vary from a slight lameness to inability to walk. The first symptoms appear in from one to three months after the deficiency begins. When both front limbs are affected the gait is stiff, and when standing, the legs may be directed slightly backward and flexed at the carpus ("knee-sprung"), or the pastern may be vertical so that the pig appears to stand on the tips of the toes. In more advanced cases the pig rests and moves about on the knees. Involvement of both hind legs, which is the rule, causes a shortening of the forward stride, lameness, and stiffness. When the lesions are well-marked the hind legs are directed forward under the body, and the tarsal joint

develops extreme flexion which becomes permanent. Posterior paralysis is one of the most frequent symptoms. Recumbency is the favorite position and the pigs are reluctant to move. Enlargements of the joints may be found in advanced types. Other frequent symptoms are fractures of the ribs, femur and vertebrae and atrophy of the muscles of the shoulder and thigh. In an excellent description of symptoms by Kernkamp[8] he mentions excessive rooting, digging, and eating of earth as if the pigs are attempting to obtain something that is lacking in the diet. In some cases the bones of the head may enlarge to such an extent that deformity is marked. Prodromal convulsions are occasional. A skin

Fig. 63.—Calcium deficiency on a diet of wheat middlings: calcium rickets. (Courtesy of S. J. Roberts.)

eruption has been described as a precursor of more definite symptoms. Dryness, thickening, and wrinkling have been observed about the eyes and over the abdomen.

In experimental pigs on a low calcium diet, in which the serum calcium fell to 5 mg. per 100 cc., the animals became typically tetanic. As described by Pedersen,[9] "they were irritable, the musculature tense, the muscular pattern, especially on the legs, very distinct, and the tail quivered incessantly. When the pigs stood in their cages they shook so the cages rattled. Both pigs had typical tetanic fits during which they, squealing, fell on the side in general spasms lasting a couple of minutes." Rapid improvement followed the administration of 1 million units vitamin D_2, and the serum calcium became normal. A daily dose of 500 units of vitamin D_2 kept animals free from the clinical symp-

toms of tetany, but the serum-calcium values, as well as the calcium absorption, were less than normal.

While the course is chronic, the prognosis is favorable and recovery is fairly prompt after correction of the diet, if deformity is not marked. Apparently this is explained by the fact that lameness and paralysis are relatively more prominent than the anatomical changes in the limbs. The prognosis is also more favorable in older, less rapidly growing pigs. With marked deformity and poor condition the case is hopeless.

Treatment.—Calcium deficiency in swine is readily corrected by feeding finely ground limestone (2 per cent of the ration). Protection against the disease is also afforded by providing access to a mixture of 9 parts bonemeal to 1 part tankage. Emphasis has been placed on the importance of vitamin D in the form of cod liver oil, or by means of exposure to sunshine, but apparently vitamin D is not essential if an adequate supply of minerals is provided. The legumes (clover or alfalfa) furnish a diet rich in calcium. In the winter diet, it is recommended that pigs receive 5 per cent ground alfalfa meal, and that sows receive 10 per cent—Bohstedt.[10] Calcium supplements are not required when the rations contain sufficient amounts, as in tankage, skimmilk, and legume hay or pasture. Less desirable forms of calcium are found in marls and dolomitic limestones. Undesirable and harmful combinations of calcium are found in rock phosphate and phosphatic limestone which contains fluorine—Mitchell,[11] Maynard.[12]

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IODINE DEFICIENCY

(Goiter; Hairless Pigs; Weak Foals; Weak Lambs)

Iodine deficiency is characterized by an enlargement of the thyroid glands (simple goiter) in newborn calves and by a hairless condition of the skin of newborn pigs. As a sporadic or endemic disease it is

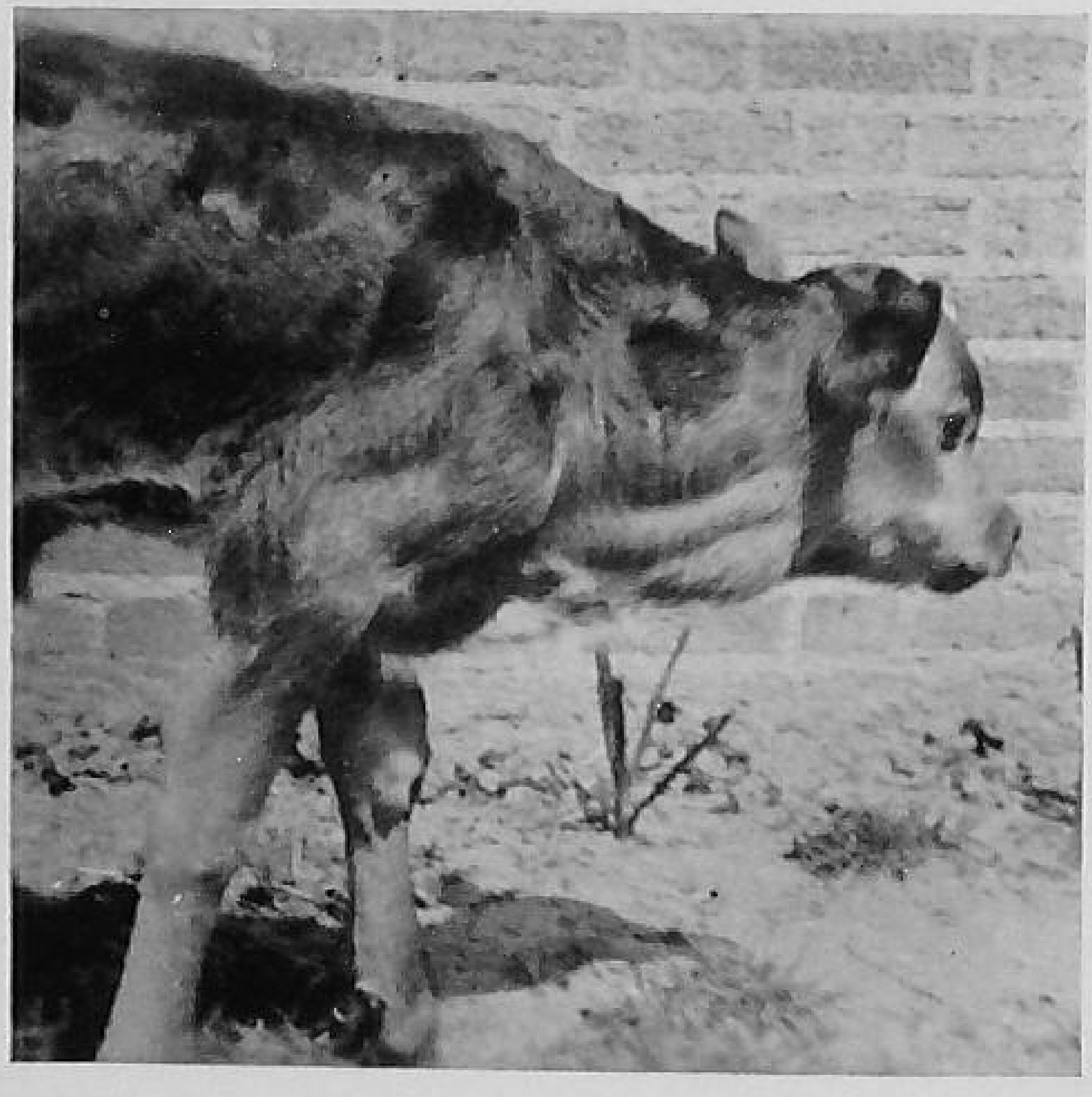

Fig. 64.—Iodine deficiency. (Photograph by W. J. Gibbons.)

widely distributed throughout the United States. It is most prevalent in this country in the region of the Great Lakes and in the Northwest. It is infrequent near the seacoast. Many cases of goiter have been reported in New York State. The disease has been described by Kalkus,[1] Welch,[2] Smith,[3] and Steenbock.[4] A deficiency of iodine in the food or water supply of the dam is the sole cause. In areas where the deficiency is extreme, as in certain parts of Washington, a goiterous swelling is frequent in mature animals, but this does not appear to affect their health. In some areas the deficiency is so slight that sporadic cases are observed only occasionally, while in others losses occur every year. In Washington it is believed that a long cold spring adds to the

severity of the disease, and that calves born in the fall are less subject to attack. There is also a cyclic variation in occurrence; this may be due to a variation of iodine content in feed and water in different seasons.

Morbid Anatomy.—There is a marked swelling of the thyroid glands, and the tissues around them are extensively infiltrated with blood and serum. The thyroids are enlarged, soft, and have the appearance of clotted blood.

Symptoms.—Calves are usually born alive, but a few are aborts. There is a marked weakness and often inability to rise. If the animal is able to stand and suckle, after being assisted to its feet, improvement may be rapid. Kalkus[1] writes that all affected calves show a marked jugular pulse. Hairless calves are rare. In swine the deficiency causes a hairless condition in newborn pigs; the majority are alive but they soon die. Often it affects only a part of the litter. Affected foals and lambs are weak, and they usually die without ever being able to stand.

Prevention.—This condition is readily prevented by giving the dam small amounts of iodine during the last three months of pregnancy. For sows, potassium iodide 1½ to 2 grains (0.09 to 0.13 Gm.) daily is sufficient. Kalkus[1] reported normal parturitions following the application of tincture of iodine to the skin. This was applied every two weeks, beginning as early as possible in pregnancy. Cows and mares received one teaspoonful and small animals half this amount applied to the skin on the inside of the flank. A convenient method of administration of iodine is to add potassium iodide to the salt—1:3500.

The need of iodine for young growing animals has not been established. The results of experimental feeding of iodine to the young have been conflicting (Iowa, Mitchell,[5] Kelly[6]).

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COPPER DEFICIENCY

(Swayback; Enzootic Ataxia; Pining)

On sand, peat, and muck soils of various countries a deficiency of

copper has seriously affected the cattle and sheep industry. In the United States this deficiency is in Florida,[1] and reports of an identical or similar disease have come from New Zealand,[2] South Africa,[3] Australia,[4] and Britain.[5]

From the work on copper deficiency in New Zealand, Cunningham concludes that "two diseases occur in stock. One disease is a simple copper deficiency which occurs in small areas and which causes enzootic ataxia in lambs, ataxia in calves, and unthriftiness and slow breeding in adult cattle.

"The second disease is peat scours, which affects cattle on most of the remaining peat land and which is most probably due to a moderate deficiency of copper, accompanied by a moderate excess of molybdenum in the fodder. The major symptoms are acute and persistent scouring affecting cattle in the spring months, debilitation of calves and cattle and reduced butterfat production. Symptoms of simple copper deficiency and of peat scours are completely controlled by supplying copper."

Symptoms.—As described by Davis et al[1] in Florida, simple copper deficiency on white and gray soil areas is characterized by retarded growth, failure to fatten, rough coarse hair which sheds freely, fading of the hair color and severe anemia in extreme cases. These symptoms occur most often in the young, but have been observed repeatedly in cows, and may develop wherever the copper content of the forage drops below 5 ppm; this involves most of the native pastures on sand soils. Simple copper deficiency does not cause the severe bone changes and diarrhea observed in the presence of molybdenum.

On peat and muck soils where the presence of molybdenum in the soil and forage accentuate the copper deficiency, and cattle tolerate less than 1 ppm of molybdenum, the symptoms are more severe. Most cases start with a severe diarrhea, followed by loss of weight, with no abnormal appetite. In cattle from 2 to 15 months of age a rickets-like condition develops in the metatarsal and metacarpal bones above the pasterns, and animals suffer from osteomalacia with fractures of the ribs, femur or humerus. Abnormal ossification of the bones in cattle on peat soil was more pronounced in cattle grazing on forage low in phosphorus. Some mature animals in good flesh and health, but depleted in both copper and phosphorus, have died suddenly under exertion before showing symptoms of chronic copper deficiency, known in New Zealand as "falling disease." Histological sections of the heart have shown muscle degeneration. Additional studies with radioactive phosphorus and molybdenum have shown that molybdenum causes a loss of phosphorus from the animal as well as a failure to utilize feed phosphorus

if copper is not supplied in the ration. In certain areas copper deficiency is further complicated by deficiency in calcium, phosphorus and iron.

Control.—Affected cattle recover rapidly when they are given a mineral supplement of copper and cobalt. When forage contains 1 to 80 ppm of molybdenum, cattle weighing 300 to 1000 pounds were protected with daily intakes of 0.5 to 2 grams of copper sulfate. Experiments have shown that cattle can retain the copper from the forage more completely while receiving cobalt. For this reason it appears that a trace of cobalt in the ration or supplement aids in the process of copper metabolism in the body. Since forages grown on nearly all of the peat soil areas have given increased yields of green forage upon application of copper to the soil, it is recommended that some copper be applied in the fertilizer. Since insufficient phosphorus intakes have aggravated copper deficiency in cattle, fertilizers on such soils should contain adequate amounts of phosphorus. In general, an 0-8-24 fertilizer containing 10 per cent of copper sulfate may be applied to peat soils at the rate of 500 pounds per acre. Two hundred to 300 pound applications annually are desirable to maintain forage yields thereafter. When fed 5 per cent of copper sulfate in the mineral supplement to cattle on peat soil, the number of animals with copper deficiency has been low. No cases of copper toxicity have been reported. A mineral drench for individual cattle that do not go to the boxes consists of 0.5 ounce of cobalt sulfate, 2.0 ounces of aluminum sulfate and 2 to 4 ounces of copper sulfate in 1 gallon of water. The dose for any animal above 300 pounds in weight is six fluid ounces once weekly for three or four weeks; inclusion of aluminum sulfate is optional. For use on white or gray mineral soils (often low in iron) this formula is used: 0.5 pound ferric ammonium citrate, 10 grams copper sulfate, and 10 grams cobalt sulfate in 1 gallon of water. Give three ounces for a calf, and six ounces for yearlings or mature animals in three successive doses at weekly intervals.

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COBALT DEFICIENCY

(Salt Sickness, Florida; Bush Sickness, New Zealand; Coast Sickness, Australia; Lake Shore Disease, Michigan; Pine Disease, Scotland; Enzootic Marasmus; Nutritional Anemia)

Cobalt deficiency is a progressive debility caused by the feeding of roughage grown on soils deficient in cobalt, or by grazing on such soils. In domestic animals it has been described only in cattle and sheep, in many separated areas. Cobalt is one of the 13 mineral elements known to be essential to the normal nutrition of animals; it belongs to the "trace" elements which are required only in minute amounts.

For many years the disease has been recognized in cattle in the northern counties of Michigan that border on Lakes Michigan and Huron—Baltzer and associates.[1] Its occurrence here is chiefly in winter and early spring followed by improvement when the stock is turned to pasture. Hay samples from affected farms contained only 0.03 to 0.06 p.p.m. of cobalt compared with a normal of 0.12. In the Coastal Plains of the Gulf and Atlantic coasts, "salt sickness" in cows is apparently due in part to cobalt deficiency, in part to iron deficiency. Cobalt deficiency has been reported from northern New York, New Hampshire,[2] Wisconsin,[3] Tennessee, and Canada.[4] In Denmark the disease was found to be much more frequent in 1945 than in previous years. In sheep in Alberta affected with an obscure form of debility, Bowstead et al.[6] observed rapid improvement following the use of 5 mg. of cobalt per head daily, while the addition of copper and iron to the diet proved detrimental. The disease has been studied extensively in Australia and New Zealand, and has been reported from Scotland—Corner and Smith.[7]

Symptoms.—As described in cattle in Michigan[8] the cows refuse to eat good alfalfa hay or cereal grain, become emaciated and in some cases die. The usual symptoms are unthriftiness, morbid appetites (pica), and rapid loss of condition after calving. The calves were apparently normal at birth, but die in from two to six weeks. The hair coat is rough, the mucous membranes pale, and there is a marked decrease or cessation of milk flow. Affected cows and calves eat filthy straw and bedding, gnaw wood, chew bones, and drink little or no water. The hemoglobin values of emaciated cows are 6 to 8 Gm. per 100 cc. as compared with a normal of 12 Gm. Tallquist hemoglobinometer readings may be as low as 40 per cent. Filmer[7] writes that there is anorexia in calves, but not in sheep, estrum is rare and abortion at 6 to 9 months is common; the pulse, respiration and temperature are normal. Death occurs in from six weeks to two years. The liver, kidneys

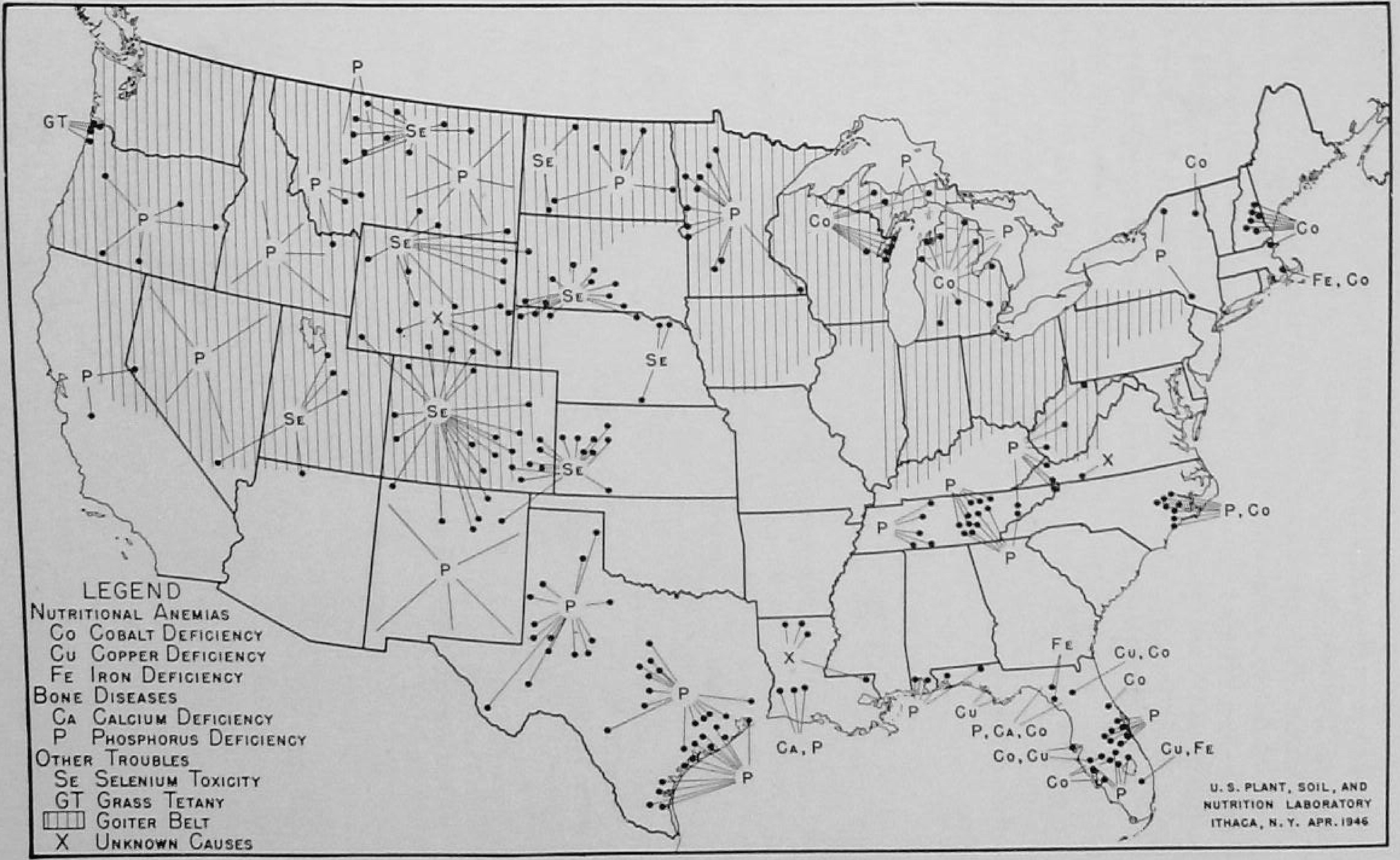

Fig. 65.—Mineral deficiencies in the United States. (Courtesy of K. C. Beeson.)

and spleen show a marked increase in iron. Diagnosis is based on the response to the administration of cobalt.

Treatment.—The curative effect of cobalt was reported by Underwood and Filmer[8] in Australia in 1935; they observed rapid improvement in sheep from a daily dose of 0.1 to 2.0 mg., and in cattle from a daily dose of 0.3 to 1.0 mg. in the form of cobaltous chloride. In Michigan a much larger dosage was prescribed by Killham.[6] One ounce (30 Gm.) of cobaltous chloride was dissolved in 1 gallon (4000 cc.) of water, and ½ ounce (15 cc.) of this solution was given daily; this was the equivalent of 2 grains (130 mg.) of cobalt. Addition of 3 Gm. cobalt sulfate or other soluble cobalt salts per ton of dairy feed is sufficient for cows; in salt mixtures add 0.5 ounce per 100 lb. of mixture.

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STIFF-LAMB DISEASE

(White Muscle Disease)

Stiff-lamb disease is a stiffness and paralysis of lambs which develops when they are turned to pasture at an age of from one to eight weeks. The lesions consist chiefly of a degeneration of the muscle fibers and bundles of the hind limbs.

Etiology.—The essential cause is unknown. For years the malady has been recognized in New York, and it has been reported from Pennsylvania, Maryland, Ohio, Michigan, Wisconsin, Montana, Oregon, Washington, and Nevada. With few exceptions the disease attacks the best lambs in a flock, and while sporadic cases may occur in the barn or yard, the majority appear soon after the flock has been turned to

pasture; as high as 10 or 15 per cent may be affected on the second day. No cases have been found in hothouse lambs, and very few in lambs born in the latter part of May or in June. Many flocks are affected repeatedly each year, while in others the attacks are only occasional.

Conditions which favor the appearance of the disease are reported by Willman and associates[1] as "A 'stiff-lamb' ration which consisted of a second cutting alfalfa hay and a mixture, by weight, of three parts whole oats, three parts whole barley and four parts of cull beans." Also "stiff-lambs have been produced at this station when clover hay and when first-cutting alfalfa hay was substituted for the second-

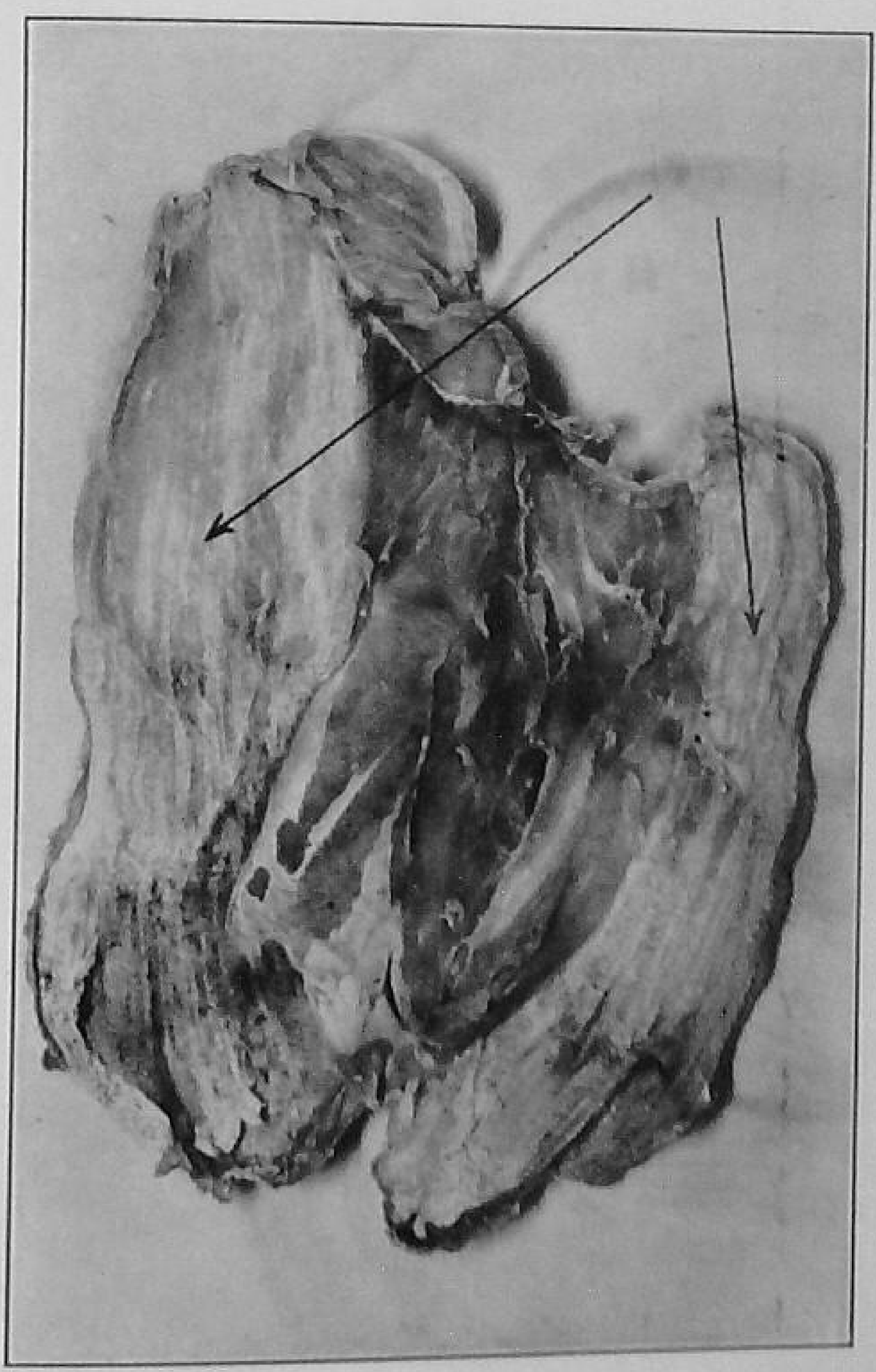

Fig. 66.—Muscular tissue of a "Stiff Lamb." Reduced one half. The whitish tissue indicated by the arrows represents the degenerated muscular tissue. This is rather an extreme case. The functional muscular tissue has undergone degeneration and much fibroplastic tissue has appeared to take the place of it. (Metzger and Hagan, Cornell Veterinarian, 1927, 17, 35.)

cutting alfalfa hay. . . . Wheat germ meal prevents the development of the disease. . . . The results of research at the Cornell Station indicate that the stiff-lamb disease is of nutritional origin. The lambs are not affected at birth. Close confinement of the pregnant ewes is not a chief causal factor. It is best to accustom lambs gradually to outdoor exercise. A large percentage of the lambs have recovered when they and their dams have been closely confined so that it was possible for the stiff lambs to nurse regularly without too great an effort." Since the administration of vitamin E prevents and cures the disease, it is highly probaable that the malady is a vitamin E deficiency.

In the ambulatory clinic, calves on an exclusive milk diet, showing symptoms of lead poisoning, and described in this text under tetany in calves (p. 377), have on autopsy shown the lesions of white muscle disease. And this condition has also been reported in calves in Nevada by Vawter and Records.[2]

Morbid Anatomy.—Characteristic postmortem lesions are found in the muscles of the affected limbs in the form of symmetrically arranged whitish streaks of muscular degeneration. As described by Metzger and Hagan,[3] "the extent of the changes varied a great deal. In the mild cases the affected muscles were noticed to be streaked with whitish lines which ran parallel with the fibers. The lines were usually narrow, one millimeter or less, but frequently were quite long. These lines sometimes were very abundant in all of the muscles of the affected part, they might occur very abundantly in certain muscles and be comparatively absent from others. . . . In more advanced cases these areas were so closely packed that portions or all of some of the muscles were whitish in color. These areas were rather dry and firmer than the normal muscles." Similar changes were sometimes found in the heart. The lesions are degenerative and not inflammatory. In the muscles of stiff lambs with no gross lesions extensive microscopic lesions may be found.

Symptoms.—At first the affected lamb shows a slight difficulty in rising and in following the ewe. This initial stiffness soon leads to distinct paralysis with inability to rise, and within three or four days, starvation or exhaustion results in death. There may be a contracture of the leg muscles, causing flexion of the digital and hock joints. The limb may easily be forced to its normal position without causing pain, but when released, it immediately returns to the flexed position. There are no general symptoms, such as depression, loss of appetite, and fever. If a paralyzed lamb is raised in a position to suckle, it takes milk actively. The muscles of the hind limbs are affected chiefly, though the attack may be limited to the front limbs, or it may be general

throughout the skeletal muscles. Recovery is infrequent. In some flocks the disease may be relatively mild, and nonfatal, but the convalescents are permanently stunted.

In the *diagnosis* one needs to consider pyemic affections in lambs which may arise from navel-ill or from wound infection after castration or docking. In these cases there may be lameness from purulent arthritis, or a paralysis of one or more limbs from a purulent meningitis. In septicemic disease of the newborn, the lamb may be unable to rise because of general weakness. A form of "stiff lamb" disease in California due to a strain of *Erysipelothrix rhusiopathiae* not pathogenic to swine has been described by Howarth.[4] The condition was a polyarthritis and the mode of infection was thought to be castration and docking wounds and navel-ill.

Treatment.—In the treatment of stiff lamb disease, dilute phosphoric acid in a dosage of 2 drams (8 cc.) three times a day for the first 24 hours and later in dram doses three times a day has been reported as curative by Schofield.[5] Willman et al.[6] (1946) write that there were no death losses in 17 stiff lambs given curative treatment with vitamin E (alpha-trocopherol), and Marsh[7] found wheat germ oil (10 cc. emulsified in 50 cc. of milk) apparently very effective when given at intervals of one to five days. In feeding experiments conducted by Willman[6] the disease was prevented by a diet for the ewe composed of mixed timothy and clover hay, corn silage and a grain mixture of two parts oats to one of bran by weight. The inclusion of liberal amounts of wheat germ meal in the rations fed to the ewes and its use in the creep-feed almost entirely prevented the disease; the amount of exercise had little or no effect on its occurrence.

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ACUTE BACTERIAL DISEASES

ANTHRAX

(Splenic Fever; Charbon)

Definition.—An acute septicemia (bacteremia) caused by *Bacillus anthracis* and characterized by enlargement of the spleen—splenic fever. All animals are susceptible.

History.—The history of anthrax is marked by three important events: it was one of the first scourges to be described in ancient and Biblical literature; its description by Koch in 1876 marks the beginning of modern bacteriology; and, when Pasteur immunized animals against anthrax in 1881, this was the first example of artificial immunization against disease by means of an attenuated culture of the specific cause of that disease.

Distribution.—Anthrax is general throughout the world, especially in Russia, Asia, Africa, the tropics and all warm climates. Heavy losses have occurred in the "old anthrax districts" of Southern California, Nebraska, South Dakota, Louisiana, Mississippi, and Texas. In "anthrax districts" the disease is most prevalent in July and August at the height of the fly season.

In the Summer of 1952, as reported in the Farm Journal for May, a ship entered New York harbor late in January with 100 tons of raw bone meal from Belgium. "Inside a month, hogs in a half dozen Midwest states were dying with anthrax—caused by eating feed containing that bone meal." The Journal of the A.V.M.A., in August, 1952, quoted the U. S. Bureau of Animal Industry of June 9, that in April and May 330 outbreaks of anthrax had occurred in 19 states; the total losses included 232 cattle, 381 swine, 79 sheep, 56 mink, and 2 mules. The suspected sources of infection were contaminated feeds—probably 280 outbreaks; infected premises—18 outbreaks; dead animals used as feed—2. In New York State Hagan[1] reports anthrax is far less prevalent than formerly, and usually involves only a single animal where it has not been known before or since; it occurs as often in the winter as in the summer, and is probably brought in in some of the commercial feeds.

In localities, known as "anthrax districts," enzootics are frequent and few states in this country are permanently free from such outbreaks. It affects chiefly herbivora—cattle, sheep, and horses, less often and less severely, swine, dogs, and chickens. It is sporadic in man as a local affection of the skin, the lungs, or the intestines; it is estimated that there are about 150 human cases each year in the United States.

Etiology.—*Bacillus Anthracis.*—The *vegetative* form of this microorganism is found in the blood of advanced clinical cases, and in the tissues of those that have recently died. It is a nonmotile rod, 2-4 microns long, arranged in short chains in the tissues and encapsulated. The organism is readily found in stained smears from the tissue or blood. Rabbits, guinea pigs, and mice are killed in from one to three days by subcutaneous or intravenous injections of infected blood. The *spore* form exists permanently in the soil. Spores originate from bacilli dropped in the excretions of either the sick or the dead. They form in the feces of the sick, and in infected districts they may also form in the feces of the well—carriers, but sporulation occurs only in the presence of air. Under favorable conditions of moisture and temperature, propagation continues in the soil. Spores are carried in hides, brushes, water, roughage, grain, vaccine, bone meal, bones, and other animal products. In 1952 contaminated bone meal was a cause of widespread infection in the United States.

Modes of Infection.—(*a*) *Soil Infection.*—Once the soil becomes infected it remains so for years. New invasions result from inundation of pastures with tannery refuse, from improper disposal of anthrax cadavers, and from flesh-eating animals and birds that carry the germs in the feces or upon the feet. Pasteur, finding spores on the surface of the ground where an anthrax cadaver had been buried, thought the earthworm brought bacilli to the surface. Spores are not found in the buried carcass and the bacillus is soon destroyed by decomposition of the tissues, so that their presence at the surface is probably explained by contamination before burial.

(*b*) *Ingestion.*—Cattle and sheep usually contract the disease from infected pastures and streams. Roughage and grain may also carry the infection and occasionally cause a sporadic stable infection. Swine, dogs, cats, and chickens sicken after eating anthrax cadavers, licking infected blood and hides, or any object that has recently been contaminated. Ingestion is favored by prolonged drought combined with extreme heat, conditions which lead to drinking of polluted water and close grazing.

(*c*) *Biting Insects.*—Horses are often inoculated in the skin from blood-sucking flies and mosquitoes, and cattle are likewise exposed. This form of infection is located on the thin-skinned ventral parts of the body and neck—sheath, mammary glands, flanks, and throat—where it causes necrosis of the skin and inflammatory edema.

(*d*) *Wound Infection.*—Contaminated hands and instruments may inoculate an operative wound in animals.

(*e*) *Vaccination Anthrax.*—In the region of the Dakotas, vaccination

with attenuated live cultures in the form of pellets injected by the farmers has been alleged to be the cause of numerous outbreaks. In explanation of this charge, Schoening[2] has expressed the view that in virulent outbreaks any biological product may respond unfavorably when used for vaccination in herds that are exposed or in the general area of a severe epidemic; that vaccination at the height of an intense outbreak of anthrax introduces some unknown factor favorable to the development of the disease, and that each repeated vaccination at brief intervals during the outbreak may be followed by deaths from anthrax. A somewhat different view of the results of vaccination with pellets has been presented by Cotton,[2] who observed that in Minnesota anthrax following their use did not occur on other farms in the near vicinity, and did not appear until after seven days following the injection.

(*f*) *Human infection* is largely an occupational disease among those who handle hides, hair, wool, and bones, or those who come in contact with anthrax cadavers, such as farmers, butchers and veterinarians. Cheap shaving brushes are an occasional source of infection. Skin infection is the most common form of anthrax in man, where it develops in the form of *malignant pustule. Intestinal anthrax* is rare in the human subject. *Inhalation anthrax* is known to occur only in man—wool sorter's disease or pulmonary anthrax.

Morbid Anatomy.—Rigor mortis fails and the carcass rapidly undergoes decomposition. The skin sometimes presents edematous swellings or necrotic patches, usually it is normal. The body openings often exude dark-red blood. Beneath the skin, in the serous and mucous membranes and in the muscles, extensive hemorrhages are frequent. Blood-stained serum is found in the body cavities. The spleen is greatly swollen, hemorrhagic, and degenerated. The liver and kidneys are swollen, congested, and soft. Hemorrhagic inflammation is nearly always present in the duodenum and in the abomasum. The blood fails to clot. The lungs are congested and the respiratory mucosa is heavily sprinkled with petechiae. While these are the characteristic changes of a typical acute case there are many exceptions. Infrequently, in peracute types, there may be no visible gross tissue changes. In general, the postmortem changes of acute septicemia and toxemia are similar, regardless of the cause.

Symptoms.—The disease occurs most frequently in summer months in pastured animals, but often it attacks horses constantly stabled when not at work, and sporadic attacks in other stabled animals are not rare. The period of incubation under natural conditions is not readily determined. After ingestion it is probably one or two weeks; after bites and wounds it may be much less.

Forms of Anthrax.—(*a*) *Peracute Anthrax* (Apoplectic).—This is always a rapidly fatal general infection. It is the usual form in sheep, a frequent form in cattle, and it is occasional in the horse. Death in convulsions may occur in from a few minutes to two or three hours. Tremors, grinding the teeth, pounding heart beat, congestion of the mucosae, dyspnea, and collapse may be noted in animals that do not drop dead without previous warning. Bloody foam is often expelled from the mouth and nostrils, and blood from the anus and vulva. This type is most prevalent in the beginning of an outbreak.

(*b*) *Acute Anthrax.*—The chief characteristics are those of a general infection ushered in with a temperature of from 105° to 107° F. The onset is sudden with marked depression, drooping of the ears, congestion and hemorrhage of the visible mucosae, muscular tremors, cessation of the milk secretion, a rapid pulse, and a high fever. As in acute septicemias, other manifestations vary according to the intensity of the invasion in different parts of the body. Thus one observes necrosis and inflammatory edema of the skin, enteritis with bloody diarrhea, excitement and mania from meningo-encephalitis, hemoglobinuria from nephritis, and swollen lymph glands. The symptoms are not the same in all individuals. Horses and cattle may continue to eat until shortly before death. The terminal symptoms are those of the peracute type with normal or subnormal temperature. The course is from one to two days, sometimes longer.

In *cattle,* swellings similar to those observed in the horse are occasional, and the course is somewhat shorter—twelve to twenty-four hours; these swellings appear at the throat, on the neck, chest, flanks, or back. But enteritis with bloody diarrhea is the usual localization. Swellings on the tongue (gloss anthrax) and the rectal mucosa have been described. The milk, if obtainable, is usually bloody.

In the *horse* the owner may have noted swelling of the mammary glands or sheath. At first this is hot and painful and within twenty-four hours it may become so extensive that walking is difficult. Yet the animal may be bright, continue to eat, and carry a normal temperature or have a fever of 106°F. Exceptionally, even after the swelling has become extensive, the temperature may fail to rise or it may go only slightly above normal. On the second day the enlargement becomes cool and painless, and the overlying skin is normal in appearance, the appetite poor, and the breathing fast, while the temperature drops to 104° or 102°. Near the end, colicky pains often appear, the temperature falls below normal, and death occurs suddenly about the third day, though it may take place much earlier. When swellings develop in the pharyngeal region—edema of the glottis, inspiratory dyspnea develops early.

Similar swellings are commonly located in the lower abdominal and thoracic regions. Less often the onset is associated with colic, enteritis, and a fatal septicemia without swellings. When anthrax appears in horses it often attacks only one or two individuals on a farm, a suggestion that it is carried by biting flies.

Swine are chiefly affected with an inflammatory edema of the throat —edema of the glottis—that soon leads to death from suffocation and septicemia. Infection is caused by eating dead carcasses, by fly bites around the ears, and in Germany by the feeding of imported bonemeal. One observes bloody froth at the lips, swelling of the throat and face, symptoms of choke, petechial hemorrhages on the skin, and the usual symptoms of an acute general infection: dullness, inappetence, seclusion from others in the herd, and a high fever. Swellings may also occur over the chest and abdomen; these may be hot to the touch but they are not painful. When infection enters through the intestines instead of the pharynx the symptoms are those of enteritis with bloody dysentery. Death usually occurs in from 12 to 36 hours, but recovery is occasional. In some instances the external lesions are absent. In the chronic form there are no distinct symptoms; lesions are found only in the abattoir. While swine are less susceptible than cattle, they are easily infected from eating contaminated refuse, and the disease in swine is limited almost entirely to farms where other species have died of anthrax. *Chickens* die within 24 hours.

(*c*) *Subacute Anthrax.*—This term applies to convalescents from the latter period of an outbreak, and to the animal that possesses a natural resistance. Since carriers have been reported in cattle and swine, one may expect occasionally to find individuals in which the disease is atypical with respect to both course and form.

(*d*) *External and Internal Anthrax.*—In man anthrax is a local infection of the skin (malignant pustule, and malignant anthrax edema), the lungs (wool sorter's disease) or the intestines. The skin form is termed external, the others internal. Malignant anthrax edema in man appears to resemble closely anthrax edema in animals.

Prognosis.—In heavily infected districts, and under exposure to swarms of flies, the disease is highly destructive to all species of domestic animals. When the infection is less abundant and virulent only a small part of a herd suffers, but the mortality in the sick is from 90 to 100 per cent. Recoveries are mainly confined to local external infections, and the last to be attacked in an epidemic. Burnett[3] has reported 192 cases of anthrax in St. Lawrence County, New York, with recoveries in 8 horses and 14 cattle.

Diagnosis.—Sudden deaths in pastured stock may be caused by

other acute infections—blackleg, hemorrhagic septicemia, by lightning stroke, and by lead or other poisoning. Cattle not infrequently die at pasture, especially in the spring, without a diagnosis being made even when the cases are thoroughly investigated. Recognition depends largely on finding the bacillus in the blood of the sick or the tissues of a fresh cadaver. Because of the importance of making an early diagnosis, smears from the blood before death are preferable. In no other disease are similar bacteria found in the circulating blood. Yet anthrax bacilli may not be found in the circulating blood until shortly before death. As a rule, however, they may be easily demonstrated from cutaneous hemorrhages immediately after death. If doubt exists a mouse or a guinea pig should be inoculated. Any acute, undiagnosed, febrile disease occurring in the summer suggests anthrax. Edematous noncrepitating swellings are highly suggestive. Autopsy lesions of special significance are: failure of rigor mortis, bloody foam at the body openings, hemorrhages throughout the body, and *enlargement of the spleen.*

For laboratory diagnosis send fresh iced blood, heart, spleen, or liver. A negative report is not always conclusive, for the bacilli die rapidly in putrefactive tissues and they may succumb in transit. A common method is to ship an ear wrapped in sterile cotton. To avoid the possibility of establishing soil infection from the cadaver, samples for diagnosis may be in the form of blood drawn by needle from the jugular vein, or one may obtain a piece of spleen through an incision between the 9th and 10th ribs. For distant shipping collect blood on swabs, blotting paper, or glass slides, and allow to dry.

Immunization.—Pasteur vaccine. The vaccine introduced by Pasteur in 1881 has been extensively employed with good results. It is the live bacillus, of two strengths, to which the subject is made immune by receiving first the weak, and twelve days later the strong. Immunity lasts approximately a year, but when the infection is virulent and massive, breaks may occur before the end of the season. It has been succeeded by those of higher efficiency. Efforts to protect against intensive exposure have led to the use of spore vaccines in series 1 to 4, in which the last has little or no attenuation.

The variety of anthrax biologics in use at the present time is somewhat confusing. In a recent report by Gochenour and associates,[4] the following are described:

Antianthrax serum produces a rapid brief immunity.

Antianthrax serm and anthrax-spore vaccine used simultaneously.

Anthrax-spore vaccine (single injection).

Anthrax-spore vaccine (2, 3 or 4 injections).

Anthrax-spore vaccine in saponin solution.
Anthrax-spore vaccine, intradermic.
Anthrax-bacillus bacterin, killed whole culture.
Anthrax-bacillus bacterin, killed washed culture.
Anthrax aggressin.

In the report presented in *Bulletin 468*,[4] upon the efficacy of anthrax biologics in producing immunity in previously unexposed animals, the following conclusion is reached:

"The immunity conferred by the living spore vaccines was especially well maintained at 300 and 360 days in the instances of the anthrax-spore vaccines (single injection) and the anthrax-spore vaccine (intradermic), and was well maintained also in the instance of the anthrax-spore vaccine in saponin solution. No appreciable immunity, however, remained in the animals vaccinated with antianthrax serum and anthrax-spore vaccine used in combination."

The prompt and effective protection of vaccination against anthrax is shown by the results obtained in sheep, as reported in *Bulletin 468:* "With anthrax-spore vaccine (intradermic) there were 100-per cent survivals at 4, 16, and 108 days, as compared with 25-per cent survivals in the control group. At 155 days there were 83-per cent survivals, as compared with 17-per cent survivals in the control group. At 300 and 360 days there were 100-per cent survivals, as compared with 33-per cent survivals in the control group.

"The biologics which produced well-marked immunity were: Anthrax bacterin (washed culture), anthrax-spore vaccine (intradermic), anthrax-spore vaccine in saponin solution, anthrax-spore vaccine (single injection), and anthrax-spore vaccine (double injection). Anthrax aggressin produced a lesser degree of immunity, and anthrax bacterin (whole culture) produced comparatively little. . . ."

Anthrax spore vaccine (intradermic) is available in four strengths according to the degree of attenuation. The weakest (No. 1) is especially indicated for horses and mules. No. 2 or 3 is commonly used for preseasonal vaccination of cattle where exposure at pasture is anticipated; this is a routine practice in anthrax districts. Anthrax spore vaccine (No. 4) in combination with 50 cc. of antianthrax serum is indicated in cattle where the disease is already present. The serum confers immunity against immediate exposure and this lasts for approximately two weeks; in the meantime the vaccine has established an active immunity which lasts throughout the season. It has been reported that in highly susceptible individuals spore vaccines may occasionally cause fatal vaccination anthrax. In anthrax districts preference is given to anthrax-spore vaccine (intradermic). Spore vac-

cines (subcutaneous) are also available and are sometimes used when this form of administration is more convenient. In the experiments reported in *Bulletin 468*, intradermic administration was more prompt and efficient than subcutaneous injections. Specially attenuated vaccines are available for sheep.

According to Boughton[5] the vaccination of sheep as a routine measure is not economically sound. But in affected herds there is a tendency for the disease to persist unless exposed animals are vaccinated which may be necessary to save the flock, and preventive vaccination is often practiced in purebred breeding flocks.

When a single animal dies on a farm where there is no previous history of anthrax, it usually is best not to vaccinate the remaining animals.[1] On noninfected farms and areas only bacterins should be used. It is possible for any of the attenuated vaccines to regain virulence and establish new centers of infection.

Sanitation.—To prevent soil contamination all cadavers should be deeply buried or burned without cutting the skin, and material that drops from the body openings or that has been in contact with virus should be destroyed by disinfection or burning. Suspected hides or animal products require thorough disinfection for the protection of both men and animals. Infected premises should be quarantined to prevent the removal of infected cattle. As far as possible the sick should be segregated and the well removed to a different pasture. Where the disease exists take the temperature of all animals night and morning and make a diagnosis of anthrax in all that have a fever.

Treatment.—Antianthrax serum has proved to be successful in the treatment of anthrax in both animals and man.[10] The dose is 100 cc., preferably in the vein. According to Derflinger,[11] an initial dose of 250 cc. is more effective. Penicillin in oil, 1 million units daily, was followed by marked improvement within 24 to 36 hours after receiving three doses—Sugg, R. S.[6] Definite and beneficial effect from the use of penicillin in the treatment of anthrax in South Africa has been reported by Wessels and DeKock.[7] Aureomycin and Terramycin have also been used in man with results comparable to those obtained from penicillin.[8] Successful treatment of vaccine anthrax with a combination of penicillin and terramycin, and terramycin alone, has been reported by Bailey.[9]

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BLACKLEG

(Black Quarter; Emphysematous Gangrene; Symptomatic Anthrax)

Definition.—An acute general infection of cattle, less frequently of sheep, caused by *Clostridium chauvoei,* and characterized by emphysematous swellings of the muscles, especially those of the gluteal region. In 1875 it was differentiated from anthrax by Bollinger. In 1879-80 Arloing, Cornevin, and Thomas described the bacillus and immunized animals with an attenuated muscle vaccine.

Etiology.—In the United States it is widespread in the Mississippi Valley, and it is a frequent enzootic in nearly every state. Distribution is world-wide. Young cattle from four months to two years of age suffer chiefly; other susceptible animals are sheep and goats, and guinea pigs by injection. It is not known to occur in horses or swine. Recovered animals are permanently immune. Purebreds are more susceptible than grades, and those in fine condition are less resistant than the poor. While the disease may occur at any season, it predominates in the spring and fall in pastured cattle, and it may occur in the winter in stabled cattle.

Clostridium chauvoei (Bacillus chauvoei).—The *vegetative* form is found in the diseased muscles of the cadaver. A small amount of this tissue injected into a guinea pig kills in from one to two days; smears made from the diaphragmatic surface of the liver of the guinea pig and stained according to Gram, or with aqueous solutions of aniline dyes, show the organism in the form of rods 3-5 microns in length, arranged singly or in pairs. Some of the rods contain spindle-shaped spores. At the point of injection one finds a local swelling, bloody infiltration of the subcutis, and sometimes gas formation. It is not pathogenic to rabbits. In parts of Europe blackleg is caused by a mixed infection of malignant edema *(Cl. septicum)* and blackleg *(Cl. chau-*

voei). In the United States the cause has been considered to be *Cl. chauvoei* only, but in 1937 Breed[1] reported that in a number of states in the Middle West, from Illinois to Colorado, heavy losses from blackleg in cattle have occurred following immunization with *Cl. chauvoei* products. On examination of tissues from these areas, *Cl. septicum* was found and in three instances *Cl. novyi* also was found. A toxin of *Cl. chauvoei* has been described by Mason.[2] Spores are formed in the tissues.

Modes of Infection.—(*a*) *Soil Infection.*—This readily occurs since spores are formed both inside and outside the cadaver. Once infected it remains so permanently.

(*b*) *Ingestion.*—Infection usually enters through the digestive tract in the form of spores from contaminated pastures or water.

(*c*) *Wound Infection.*—Earlier writers accepted the view that blackleg is chiefly acquired through small wounds in the skin or mucosa, and this view is still held by some. Heller[3] writes that "the mode of infection in blackleg, beyond its being an occasional contamination of gross wounds, is entirely unknown." Blackleg in lambs following vaccination for enterotoxemia has been reported by Albiston[4] of Australia.

Morbid Anatomy.—Putrefaction develops rapidly except in the affected muscles, so that extreme bloat is present shortly after death. In the affected muscles gas formation continues in the cadaver, leads to marked tympany and causes the legs on the upper side to extend straight out. Bloody froth often exudes from the mouth, nostrils, and anus. The skin over the swellings is usually normal but in the center it may have undergone dry gangrene. On incision the connective tissue under the skin and between the muscles is found to be infiltrated with discolored bloody serum and gas. This emanates an odor that is usually described as sweetish-sour or like that of rancid butter. The muscles are swollen, emphysematous, dark or black, and friable. Swelling is due to distension with gas that presses the bundles apart, prevents collapse when they are cut, and gives to them a dry spongy texture. Similar swellings are found rarely in the masseter muscles, the tongue, pharynx, diaphragm, and, according to Law[5] in the pleura, lungs, and even in the walls of the stomach or intestines.

On opening the abdominal cavity one may find serum, hemorrhage, fibrinous exudate on the peritoneum, and enteritis. The spleen is usually normal but it may be swollen and hemorrhagic. The liver is swollen and congested; it may contain circumscribed dry yellowish foci from ¼ to 1 inch in diameter. The kidney changes are similar to those in the liver. In the chest cavity one finds changes similar to those in the abdomen: bloody serum, hemorrhage, and fibrinous exudate on the

serosa, degeneration of the heart muscle, and gelatinous infiltration of the interlobular lung tissue. According to Ravenna[6] endocarditis is frequent in calves. The blood, except in the swellings, is normal and coagulates readily.

Symptoms.—The incubation period is from one to five days. The onset is sudden. The initial symptom is usually lameness with or without fever, or there may only be signs of a marked general infection—depression, fever tremors.

Swellings are the most characteristic symptoms; these develop in the muscles of the shoulder, hip, chest, back or flank, less frequently in those of the neck, pharynx or tongue, and never below the carpal or tarsal joints or on the tail. The swelling is at first small, hot, and painful. In a few hours it is extensive, *crepitating,* and less sensitive, and soon it is cool and painless. Finally the skin over the center may become dark, dry, and parchment-like in consistency. Percussion reveals tympany from the collection of gas beneath. Incision releases a frothy, dark, sour-smelling fluid. The connective tissue adjacent to the swelling undergoes edematous infiltration and the neighboring lymph glands are swollen. The *general symptoms* are complete anorexia, marked prostration, muscular tremors, congested mucosae, dyspnea, rapid pulse, and a high fever that becomes subnormal shortly before death. Colic may be present. In most cases the disease is fatal in from twelve to forty-eight hours. A milder form with recovery is sometimes observed in older animals.

Diagnosis.—The appearance in young pastured cattle of a rapidly fatal febrile disease associated with crepitating swellings in the heavily muscled parts is sufficient evidence to justify a diagnosis of blackleg. In its usual typical form it is not liable to be confused with other diseases. In the few cases of blackleg encountered in our ambulatory clinic the initial case has occurred in February or March in stabled young stock; in one of these, swellings and fever were absent and diagnosis was made by finding the typical changes in the muscles on autopsy.

Sweet clover poisoning has been mistaken for blackleg because of the lameness and skin swellings, but the swellings of sweet clover poisonings do not crepitate. In *anthrax* the spleen is usually enlarged, the blood fails to clot, and if swellings are present they do not crepitate. The anthrax bacillus is found in the circulating blood shortly before death, or in subcutaneous hemorrhages directly after death. *Hemorrhagic septicemia* affects animals of all ages; swellings are seldom present, but they may be found in the pharyngeal region; severe involvement of the lungs is the rule.

Since any of the three infections, anthrax, hemorrhagic septicemia,

or blackleg, may assume a purely septicemic form, differential diagnosis may depend upon microscopic examination of the smears, inoculation of small animals, or cultures. The lesions produced in guinea pigs by *Cl. chauvoei* are characteristic—inflammatory edema at the point of inoculation, and spore-containing, single or paired rods in smears taken from the peritoneum or diaphragmatic surface of the liver. Ravenna[6] has reported that in the absence of local swellings other characteristic changes are usually present in blackleg in the form of an endocarditis of the right heart, an endoarteritis of the pulmonary artery, or inflammatory changes in the heart muscle.

Infection with *Cl. septicum* (malignant edema) is stated by Heller[3] to be an occasional cause of gas phlegmon in cattle, and the usual cause of braxy in sheep; while in horses and swine gas phlegmons are caused by *Cl. septicum* but not by *Cl. chauvoei*. According to Moore and Hagan,[7] "differentiation of blackleg and malignant-edema infections of herbivora is not always easy; most of the cases are diagnosed as blackleg." Meyer[8] has written that "because of the frequent absence of typical swellings the disease is not so easily recognized as it appears from the text books."

Prophylaxis.—*Immunization.*—Losses from blackleg may be readily prevented by vaccination.

(*a*) *Arloing's Vaccine.*—This was a double vaccine first made in France in 1883 from dried pulverized diseased muscles attenuated by heat at 100° to 104° C., and 90° to 94° C. The weaker was injected first in the subcutis of the tail, followed in ten days by the stronger. *Kitt's modification,* 1888, was from the same kind of material heated for six hours at 85° to 90° C. It was used as a single injection in the subcutis of the shoulder region. In 1896 Nörgaard developed a single injection vaccine heated for six days at 94° to 95° C. This was distributed free by the United States Bureau of Animal Industry until 1922, and it proved to be highly effective. While these vaccines were of great value, they contained the living spores which sometimes caused the disease in vaccinated stock.

(*b*) *Blackleg Aggressin.*—This is a germ-free filtrate from diseased tissues of a blackleg carcass. Schöbl,[9] applying Bail's aggressin theory, immunized guinea pigs in 1911 and cattle in 1912 by this method. It produces an active immunity that is sometimes claimed to be permanent. Field trials conducted by Hart[10] in California "showed that this material will not confer life immunity in animals vaccinated at 6 months of age. . . . It is, therefore, recommended that where this material (blackleg culture filtrate) is used, all animals of susceptible age be vaccinated twice yearly, the same as has been the practice with the

old muscle vaccine." Mohler[11] states that "the immunity conferred by the vaccination varies according to the vaccine used; aggressin and filtrate apparently confer the longer immunity which may last for 18 months or longer. Animals that are vaccinated with powder vaccine before they are 6 months old and those in badly infected districts should be revaccinated before the next blackleg season." Blackleg aggressin has been widely and successfully used in the United States.

(c) *Blackleg Filtrate.*—A germ-free filtrate from cultures of blackleg organism in a special media containing liver and other tissues. It is an artificial aggressin developed by Nitta[12] in Japan. Its action is that of natural aggressin and the cost of production is less.

(d) *Sterile Cultures.*—Leclainche and Vallée[13] have reported that young cultures of *Cl. chauvoei* rendered sterile by the addition of formol become avirulent and nontoxic and yield sterile antigens that are far more active in the production of immunity than filtered cultures. These bacterins (anacultures) are now widely used in the United States; they are effective, safe, and relatively inexpensive. Because of the frequency of *Cl. septicum* infection in blackleg, especially in sheep, a bacterin containing both *Cl. chauvoei* and *septicum* is often used.

(e) *Blackleg Serum.*—In existing outbreaks the well may be promptly immunized for fifteen to thirty days with 30 to 60 cc. of anti-blackleg serum. Vaccine may be given two weeks after the injection of serum.

Heller[3] states that animals immunized against the vibrion-septique group are not immunized against blackleg and vice versa and expresses the opinion that cattle should be immunized against both groups, but perhaps such practice should depend on the nature of the organism found to infect a particular locality.

Sanitation.—Blackleg cadavers are the chief source of soil infection. They harbor the germs in large numbers and liberate them from both artificial and natural body openings. For this reason every dead animal should be promptly burned or buried; and with few exceptions the sick should be disposed of in the same manner. Disinfect all woodwork or utensils that have come in contact with the infection. The surface of the ground may be made safe by burning it over with a heavy layer of straw. Because of the vitality of the spores of blackleg, pastures may remain infected for years, even when kept free of cattle. In regions where contamination of the soil is limited the disease may be avoided by a change of pasture. It seems probable that in time an infected pasture may become free of the germs if all animals feeding upon it are vaccinated in order to prevent the addition of new infection from blackleg cadavers.

Treatment.—Blackleg serum is indicated for the sick. Its value is limited largely to those animals in a diseased herd that are found to be carrying a fever on routine examination of apparently normal individuals. The usual dose is 100 to 200 cc., though twice this amount may be given. Penicillin alone, or combined with sulfamerazine, or with antiblackleg serum has been effective.[14]

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MALIGNANT EDEMA

(Gas Phlegmon; Braxy in Sheep)

Malignant edema is a rapidly fatal gas phlegmon caused by various strains of *Clostridium septicum*. It is not infrequent in the United States.

In the *horse* it is a wound infection disease caused by a nail or splinter that enters where there is an abundance of fascia and few muscles, such as the face or limbs. In *swine* it sometimes follows castration or vaccination when the animals are kept in filthy yards or pens. In *sheep* it is a wound infection disease after castration, docking,

or shearing, and in a similar manner it may develop in *cattle* after castration. Within a few hours to a few days after the injury, crepitating swellings appear and the wound exudes a dirty reddish thin exudate. The temperature is high and toxic symptoms are marked. In wounds of the face of the horse the swelling soon spreads to the entire head and neck, often to the lungs, causing death in from 24 to 48 hours. The temperature is high and toxic symptoms are marked. Severe purpura hemorrhagica affecting the head presents similar symptoms, but there is no creptitation.

On *autopsy* the incised gangrenous swellings exude a dirty reddish exudate mixed with gas bubbles. With the exception of a marked edema of the lungs the internal changes are like those found in other acute septic conditions.

Braxy.—*Cl. septicum* is also regarded as the cause of *braxy*, a disease of the digestive tract of sheep in certain areas in Great Britain, and to some extent in other countries. As described by Gaiger,[1] braxy in Scotland is chiefly a diffuse intense inflammation of the abomasum affecting young animals in the late fall and winter. The course is so rapidly fatal that affected lambs are seldom seen alive. Gaiger believes that frosted grass in the rumen is a contributory influence. He states that the disease is more of the nature of a wound infection, a sort of "gas gangrene" of the stomach with an organism of the *Clostridium septicum* type as the sole invader, and that if sheep live through the first winter on infected land they are immune for life. M'Ewen[2] mentions two conditions in the guinea pig which serve to characterize this infection: (1) the presence of the organism in chains in smears from the surface of the liver, and (2) intense infection of the stomach and the small intestines, especially the latter. As described in braxy in sheep, *Cl. septicum* is a motile anerobe similar in morphology to the blackleg bacillus. In the cadaver it is found in the wall of the abomasum, the peritoneal fluid, and the heart blood. It kills guinea pigs in 12-24 hours and causes lesions similar to those of blackleg. Rabbits are less susceptible.

Morbid Anatomy.—Sheep that have acquired the disease at pasture, with no visible wound infection, show marked deep inflammation of the abomasum, sometimes with edema. This is the primary lesion; there may be absence of other gross lesions. There is a slight excess of peritoneal fluid. Gas phlegmon may be present in the muscles, presenting lesions identical in appearance with those of blackleg.

Symptoms.—The onset is sudden and marked by anorexia, dullness, and a fever of 106°-108° F. Death occurs within a few hours. Localization of gas swellings in muscles is frequent. A slight or marked disten-

sion of the abdomen is common. The disease may be prevented by double vaccination with formalized cultures. Previous to vaccination the losses were sometimes from 20 to 30 per cent of a flock. Filtrates are of comparatively less value than in blackleg.

Gas Phlegmon of Swine is met with occasionally as a result of accidental wound infection, and after immunization against hog cholera under filthy conditions. Here, also, the cause is *Cl. septicum*. The disease has been described in the United States by K. F. Meyer.[3]

Malignant Edema of Cattle is reported frequently from Europe, where it is considered to be a parturient infection. Fincher[4] has described one typical case in this country due to wound infection, and to judge by unpublished reports the disease in cattle is not rare in the United States. An excellent report on gas edema diseases has been made by Scott and associates.[5]

Immunization against *Cl. septicum* infection is obtained by the use of bacterins (anacultures), as in blackleg.

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BACILLARY HEMOGLOBINURIA OF CATTLE AND SHEEP

Bacillary hemoglobinuria is defined by Vawter[1] as a peracute infectious malady characterized by high fever, depression, rapid hemolysis, hemoglobinuria, and bowel hemorrhages, terminating in death in 24 to 36 hours. The infective germ is *Clostridium hemolyticum*. The most characteristic lesion is a large infarct 5 to 20 centimeters in diameter which is always found in the liver of affected cattle or sheep.

The disease is endemic in swampy poorly drained mountain valleys of Nevada, California and Oregon. It has been diagnosed in Idaho, Montana, Louisiana, Utah and Texas, and it has also been observed in Mexico and Chile. Seasonal occurrence is chiefly from June to November, with sporadic cases through the winter. In infected areas susceptible additions come down in rapid succession in a week to ten days, and losses may run from 10 to 25 per cent in non-vaccinated

cattle. The postmortem changes are generalized icterus, extensive subcutaneous and subserous hemorrhages, and the presence of a large infarct in the liver. The body cavities contain bloody serum. The onset is sudden with loss of appetite, rumination, and milk flow. The eyes are sunken and the mucous membranes are congested or yellow. The temperature at first may reach 106° F., but it soon falls. The bowel evacuations are at first scanty, and bloody diarrhea may appear later. The urine is dark red (hemoglobinuria) and foamy. The red cell count ranges from 1.5 to 4.5 million, and the hemoglobin from 3.5 to 8 Gm. per 100 cc. The disease may be confused with anthrax because of the peracute attack, the sudden appearance of dark-red urine, and the similarity of the postmorten changes. The most important difference is the anemic infarct of the liver which is always present in bacillary hemoglobinuria of cattle and sheep. The average course is about 36 hours and the mortality in untreated animals is over 95 per cent. When given early, antitoxic serum (500 cc. per vein) is often followed by rapid recovery. Immunization for a period of one year is obtained from a formalin-killed toxoid bacterin.

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BLACK DISEASE

(Infectious Necrotic Hepatitis)

Definition.—An acute, infectious, highly fatal toxemic disease of sheep, and occasionally of cattle, following the multiplication of previously latent spores of *Cl. novyi* type B *(Cl. edematiens; B. gigas)* in areas of liver necrosis due to injury from liver flukes.

The cause of this disease was first identified in 1927 by Turner and Davesné[1] in Australia. It was first recognized in the United States by Marsh[2] in Montana, where the losses have been so great that many owners have gone out of the sheep business. In 1939 it was reported by Shaw et al.[3] as a hitherto widespread disease of Oregon sheep occurring in fluky pastures. The causal organism exists in the soil of affected districts, and within the animal it is usually confined to the hepatic necrotic foci. In areas in which liver fluke exists severe losses among sheep occur which are not directly due to liver infestation, but are the result of bacterial activity secondary to fluke infestation. Death is produced by a toxin elaborated by *Cl. novyi* in fluke-infested livers.

On *autopsy* there are many subcutaneous hemorrhages on the back and sides from which the name "black disease" has been derived in Australia. The pericardium is distended with clear fluid. On section of the liver from one to several yellowish necrotic foci one to several centimeters in diameter are found (Turner[4]) and these are the most characteristic lesions of the disease.

There are no well-defined symptoms; often the animal is found dead. It has been confused with hemorrhagic septicemia and braxy. The single vaccination with a 5 cc. dose of alum precipitated toxoid immunizes sheep against *Cl. novyi* for at least 17 months (Tunnicliff[5]).

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HEMORRHAGIC SEPTICEMIA OF CATTLE

(Pasteurellosis bovis; Shipping Fever; Stock-Yards Pneumonia)

Definition.—Hemorrhagic septicemia is the name of a group of infectious diseases, usually acute, caused by *Pasteurella multocida*, occurring in cattle, sheep, swine, birds, and rabbits, and characterized by hemorrhages in the internal organs.

In cattle it is an acute general infection of sudden onset, high fever, and pneumonia, less often by gastroenteritis, or only by extensive hemorrhages of the internal organs. A form associated with inflammatory edema of the skin (edematous form) has also been described.

Etiology.—Bollinger,[1] in 1878, first described the disease among wild boar, deer, and cattle in Munich.

According to Washburn[2] hemorrhagic septicemia appeared in cattle and deer in Texas in 1896. It was reported from Tennessee by Fennimore[3] in 1898, and from Minnesota by Reynolds,[4] and Brimhall[5] in 1902-1904. While natural infection between cattle occurs readily, artificial inoculation of cattle with cultures of *P. boviseptica* is usually without effect. In 1912, however, Mohler and Eichhorn[6] reported recovery of a strain of *Pasteurella* from buffalo in Yellowstone Park

which was pathogenic to cattle when injected subcutaneously. The buffalo strain, *P. bubaliseptica,* is the most virulent member of the group and it is used in the preparation of most commercial bacterins.

Because of the presence of avirulent strains of *Pasteurella* in the tissues of normal animals, as well as in the tissues of animals that have died of hemorrhagic septicemia in a typical form, there is doubt in the minds of some that *Pasteurella* is the cause of the disease. Murray[7] states that in Iowa there are many farms where the disease has been known to appear periodically for many years. Buckley and Gochenour,[8] writing of the group, report that it occurs frequently and disastrously. Moore and McAuliff[9] have reported its occurrence in about fifty dairies in Cortland County, New York. Jones and Little[10] attributed an outbreak of pneumonia in dairy cows in New Jersey to *Bacillus bovisepticus,* and they were able to transmit the disease to calves. The disease is frequent in the United States, both as an endemic and a sporadic affection, but the chief losses are in recently shipped cattle, or in herds to which such have been added. In recent years there has been an increase in the incidence of hemorrhagic septicemia in the northeastern part of the United States. It is now widely prevalent in areas where it was unknown ten or twenty years ago; it has become a serious disease of dairy cattle in the East and it is reported to be increasing in the Middle West. Outbreaks are not infrequent on farms where there have been no recent additions, and no unusual exposure. The first outbreak of this disease in cattle in Norway to be officially recognized was described by Hellesnes[11] in 1936. The causal organism was closely related to that which causes pasteurellosis in reindeer. Transmission experiments in two cows were successful; in one an intratracheal inoculation caused the pectoral form, and a subcutaneous inoculation caused the edematous form.

When the resistance of an animal is lowered, as from transportation, there is an increased susceptibility to an attack. When animals that have been transported are added to a herd, the permanent members of the herd may be the only ones to acquire the disease. In such animals there is no evidence of a lowered resistance; the newly added animals have acted as carriers of an infection capable of causing disease in the absence of any predisposing influence.

Bacteriology.—The organism of the group *Pasteurella* is a polymorphic aerobe. It is a nonmotile gram-negative rod and forms no spores. In smears taken from diseased tissues, it gives, singly or in short chains, a bipolar stain with aniline dyes. Guinea pigs or rabbits injected subcutaneously with material from the lesions soon die, though such material from cattle may not be fatal to rabbits. Its habitat is

the upper respiratory tract and the digestive tract of normal animals. It is a saprophyte and a faculative pathogenic parasite that succumbs quickly to the direct action of mild disinfectants. Its virulence is extremely variable, often depending on a marked lowering of the vitality of the affected animal—fatigue from transportation ("shipping fever"). According to Priestly,[12] virulence depends on the presence of a capsule and only a virulent capsulated strain should be used for immunization. The virulence also varies according to the species. Apparently *P. bubaliseptica* exceeds all others in this respect. There are many reports of the disease in water buffalo in warm climates, where the infection is highly fatal. In this species the disease is termed barbone. In 1883 Kitt proved the bipolar bacillus to be the cause of the disease described by Bollinger. In 1880 Pasteur found it to be the cause of chicken cholera; in 1881 Gaffky found it to be the cause of rabbit septicemia; and in 1886 Loeffler found it to be the cause of swine plague. In 1886, Hueppe,[13] recognizing that the causes of these diseases were identical so far as could then be determined by animal inoculation, gave the name hemorrhagic septicemia to the entire group. These microorganisms are still classified as a single species under the name *Pasteurella multocida (pleuriseptica)*, and also as a multiple species according to their source as *Pasteurella boviseptica* in cattle, *P. suiseptica* in swine, *P. oviseptica* in sheep, *P. aviseptica* in fowls, etc. The convenience of a flexible classification, however confusing, is shown in a report from Jones[14] (1921) who writes, "it has been possible to show that certain well-defined cultural and serological groups exist among the strains of what may be termed the bovisepticus group." Since the infection does not spread from one species to another, the need for a multiple clinical terminology is obvious.

Morbid Anatomy.—The unopened cadaver is negative except in the *edematous* form where one finds subcutaneous edema of the head, neck, and throat, gelatinous infiltration around the larynx and pharynx, and swelling of the adjacent lymph glands.

In the *pulmonary* form the body cavities often contain quantities of fluid. Extensive pleuritic adhesions are sometimes present. The lungs present extensive consolidation. On section these variously colored pneumonic areas are separated by a marked edematous thickening of the interlobular septa; this gives to the lung a typical and striking marbled appearance. When the course has been somewhat prolonged, necrosis and abscess formation are found. The bronchi are congested or hemorrhagic and contain exudate. Pulmonary emphysema may be the only distinct lesion present. The general *septicemic lesions* may be slight or extensive; they include hemorrhages in the subcutis and intermus-

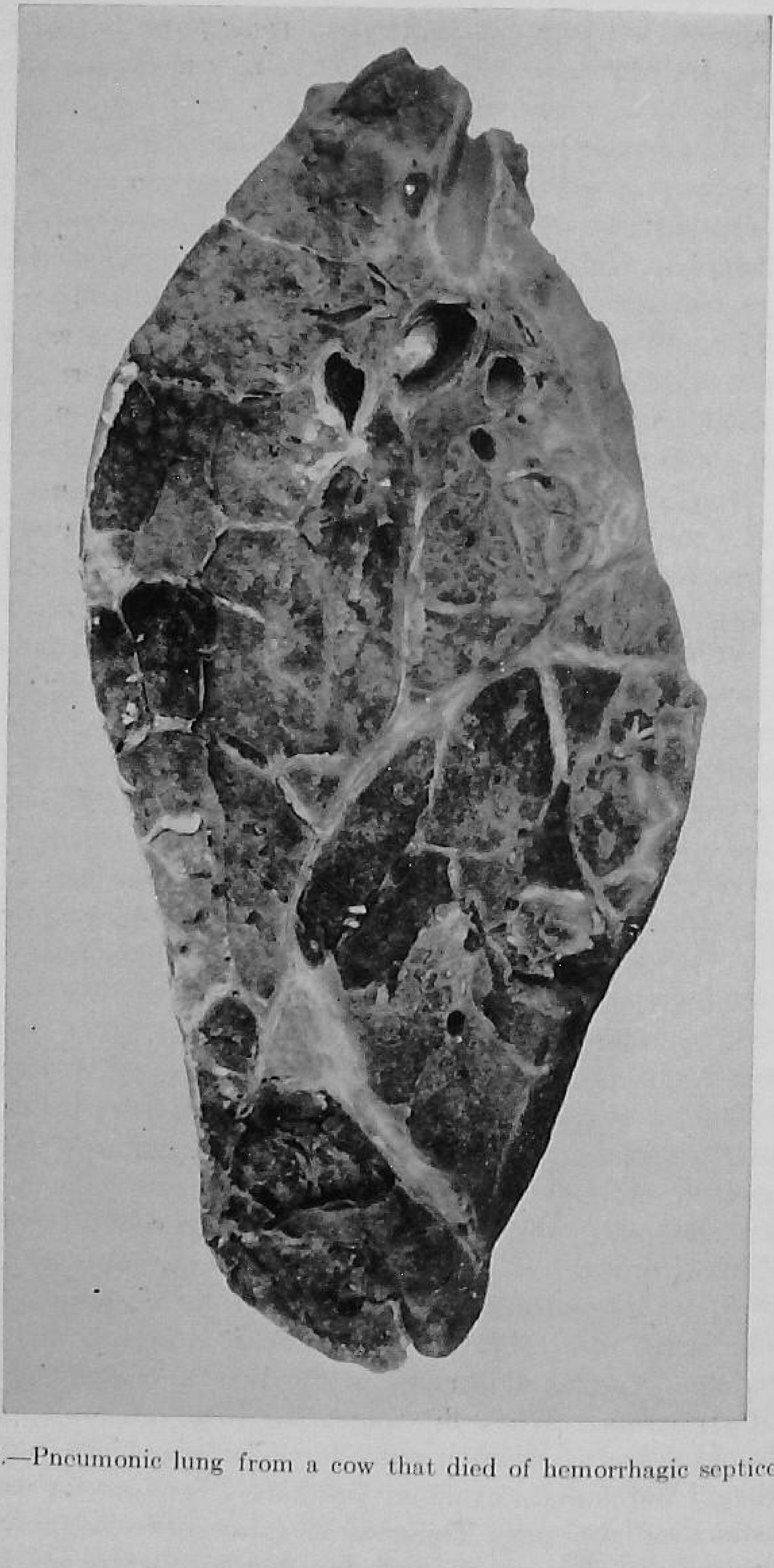

Fig. 67.—Pneumonic lung from a cow that died of hemorrhagic septicemia.

cular tissue, swelling and hemorrhage of the lymph glands, hemorrhage on the peritoneum and pleura, and hemorrhagic gastroenteritis. Often the pericardium is covered with hemorrhages and contains reddish serum.

In typical endemic pneumonia of calves whose lungs yield *Pasteurella* in pure culture, the pneumonic lesions often are unlike those described as characteristic of hemorrhagic septicemia. Edema of the interlobular tissue may not be present, and there may be no marbling of the cut surface. On the cut surface one observes red and gray hepatization with small reddish or grayish areas. The appearance is not unlike that of a streptococcic pneumonia.

Symptoms.—Pneumonic Form.—This is the most common type in the northeastern part of the United States where usually it is a stable disease. As a rule the infection is introduced by animals that have recently been shipped. Thus it is brought to New York by cows that have suffered from fatigue and exposure during shipment from the West. The new additions may be sick on arrival, they may sicken later, or they may remain well and serve only as carriers. Heavy losses sometimes result when cattle are assembled for exhibition or sales, or when such animals are returned to the farms. The most destructive outbreaks of this kind occur in the late fall and in the winter. Occasionally the disease attacks animals in a stabled herd to which no additions have been made, and it may appear in pastured stock.

The period of incubation is from 2 to 5 days. The onset is usually sudden. On the first day one or more cows are taken with a bloody diarrhea, pneumonia, and fever. On the second and third days it extends to others, either in a severe or a mild form. One or more of the first to sicken often die within the first twenty-four hours. Often the deaths all occur within the first two or three days. Exceptionally animals continue to die during the entire course of the outbreak. The mortality is especially high in cows that have recently freshened. Thermometer readings of the herd may reveal several cases that appear to be well yet carry a high fever.

The characteristic symptoms are prostration, drooping ears, congested or hemorrhagic mucosae, lachrymation, fast pulse, rapid breathing, and a temperature of from 104° to 106° F. with chills; in some cases the temperature remains normal. Anorexia is complete and the milk flow ceases. Extreme dyspnea is marked by extended head, open mouth, and protruded tongue. With each breath is an expiratory grunt. Cough and induced cough are usually present. Examination of the chest reveals râles, soreness on percussion, and possibly areas of dullness. Bloody diarrhea is common, though constipation may occur, and hema-

turia has been observed. The first to be taken often die quickly, while others recover after a variable course. As in all acute septicemias, lesions may develop in the brain, causing mania and other symptoms of encephalitis. Or such symptoms may be due to the presence of toxins that fail to cause visible lesions—meningismus.

Edematous Form.—In the edematous form described by Moore and McAuliff,[9] they mention urticaria-like swellings about the eyes and flanks, and a severe conjunctivitis with small hemorrhages in the mucosae. Edematous swellings may also develop in the region of the pharynx, and around the anus and vulva, or they may extend from the limbs to the body. The general symptoms are those of the septicemic form.

Acute Septicemic Type.—This form presents the characteristics that are common to the acute fatal septicemias without special localization, as anthrax and blackleg. It sets in rapidly and often leads to death in from 12 to 24 hours.

A *cerebral* and an *intestinal* form have been described, but regardless of where the disease may localize, be it the meninges, the intestines, the lungs, or the subcutis, the distinguishing features in well-marked attacks are those of a septicemia. A *chronic* form has also been described, (Hutyra); the subjects had pneumonia and diarrhea and were emaciated.

Hemorrhagic septicemia is thought by some to be the most common infection in calf pneumonia. Jones and Little[15] have described an outbreak in which some of the calves had pneumonia while others suffered only from nasal catarrh. From the latter they obtained cultures of *bovisepticus*, Group I. The disease was reproduced experimentally by brushing the nasal mucosa of other calves with swabs from cultures.

Diagnosis.—A diagnosis of hemorrhagic septicemia invites controversy for the following reasons: First, the normal habitat of the bacillus is on the mucosae of well animals, so that its presence in the tissues of the dead is not conclusive evidence that it caused the disease. Second, a bacteriological examination of animals that have died of the disease may be negative. Third, the name has often been applied loosely to obscure conditions and to diseases of another nature; this has led some to doubt that the bacillus is ever pathogenic, and to interpret a diagnosis of hemorrhagic septicemia as evidence of the veterinarian's ignorance. Regardless of these limitations, the disease is a reality that often appears as a well-recognized and disastrous attack.

The usual form of "stock-yards pneumonia," occurs chiefly after new animals have been added to the herd; it is mainly a stable infection and it shows no age selection.

When the septicemic form occurs in pastured stock without definite localization it is not easily differentiated from similar forms of anthrax or blackleg. A differential suggestion may come from a consideration of former endemics in the same locality and the age of the victims. *P. bovisepticus* is usually present in the diseased tissues, but bacteriologic examinations are more conclusive for anthrax and blackleg than for this disease. Cattle are frequently poisoned by lead and nitrate of soda; these cause sudden deaths with lesions resembling those of septicemia.

Prophylaxis.—Vaccination against hemorrhagic septicemia in bovines was first practiced in the United States by Mohler and Eichhorn;[6] they used a double attenuated vaccine in the buffalo in Yellowstone Park. Hardenbergh and Boerner[16] later reported the use of live cultures, but the use of these has not become general. In 1924 Buckley and Gochenour[8] wrote, "With a number of carefully controlled experiments we have proved that susceptible animals can be actively immunized against hemorrhagic septicemia with bacterins, vaccines, and aggressins. The immunity conferred by these products will persist for one year and maybe longer. Probably the most important consideration in a production of active immunity is the time required to produce such an immunity. A series of experiments conducted on cattle and also on small laboratory animals has shown that vaccinated cattle are more susceptible to the disease for the first day or two than unvaccinated animals. Also, that increased resistance does not become demonstrable until about the sixth to the ninth day following vaccination. Between the ninth and fourteenth days the resistance to hemorrhagic septicemia becomes so fully established that the treated animals are able to withstand very severe exposures. Several hundred fatal doses of virus do not inconvenience animals that have been vaccinated fourteen days or more previously." Buckley and Gochenour[8] reported in 1924 that aggressins afforded greater protection than do vaccines or bacterins. The dose of aggressin for cattle is 5 cc. for bovines, use only aggressin prepared from bovine tissues, and for equines use only that prepared from equine tissues. Severe anaphylaxis and even fatalities have occurred in cattle from repeated injection of an aggressin prepared from tissues of another species. In the vaccination of animals previous to going on the show circuit we have observed that the use of aggressins before leaving the farm has apparently afforded complete protection.

Because of the time required to produce active immunity aggressins and bacterins should be given at least ten days before danger of exposure. Their use is not recommended in exposed or infected herds, in

animals intended for immediate shipment, or in those that have recently been in transit. For purebred and other valuable cattle that are to be shipped immediately, protection may be obtained by the use of anti-hemorrhagic septicemia serum (50 cc. before shipment and from 30 to 50 cc. on arrival at destination). Gibbons and Fincher[17] have reported various complications following the administration of aggressins and bacterins. Among these were urticaria, colic, edema, and dyspnea following aggressin; and depression, trembling, extreme dyspnea, and even death after injection of a bacterin. The reactions following injections of bacterins may possibly be due to the use of serum, and possibly horse serum, in whole culture broth products.

The result of vaccination in dealer herds may be unsatisfactory. Following such vaccination, even when there have been no known recent additions, the disease may appear a week later in an epidemic form. Such experiences are difficult to explain and they are also embarrassing.

In outbreaks of calf pneumonia I have never been able to observe any benefit from serum, and in such epidemics calves previously vaccinated with bacterin and those vaccinated with aggressin have subsequently come down with the disease at the same rate as nonvaccinated calves.

Treatment.—Anti-hemorrhagic septicemia serum (100 cc. to 250 cc. daily) is an effective remedy for cattle that are sick. When the disease first appears and only two or three cows are affected, it is reasonably certain that others will be found with a high fever within twenty-four hours. If a bacterin or an aggressin is given to the apparently well cows on the first call, this will be given to a few animals when the infection is in the late stage of incubation and the cows are about to come down with the disease. If the first effect of either a bacterin or an aggressin is to increase the susceptibility of infection, as reported by Buckley and Gochenour,[8] their use in the beginning of an outbreak in a herd should contribute to its spread. There is no logical reason for the use of either bacterins or aggressins in such cases. It is more logical to administer maximum doses of serum to the sick and smaller amounts (50 cc.) to the well. The protective action of serum cannot be depended on for more than four days. Take the temperatures of all animals in the herd two or three times daily and when possible separate the well from the sick.

Regardless of the fact that Buckley and Gochenour[8] found increased suceptibility the first day or two following vaccination, and increased resistance only after the sixth to the ninth day, some veterinarians believe that either bacterins or aggressins are useful in both sick and well animals in infected herds. There is no rational basis for such medi-

cation and it is improbable that time and experience will justfy the method; it is possible that apparent improvement in such cases is due to the natural resistance of the animals. There are numerous examples of a high mortality from hemorrhagic septicemia after injection of bacterins in herds where the disease already existed.

The symptomatic treatment of hemorrhagic septicemia is like that of pneumonia, page 46. Calcium gluconate and camphorated oil are beneficial; where the use of serum is prohibitive because of the cost, one may depend upon this combination alone. The sulfonamides are widely used. These are given orally in doses of from ¾ to 1½ grains per pound body weight (0.09 to 0.18 Gm. per kilo) the first day. Reduce the dosage 25 per cent the second day and 50 per cent the third day. Administer in divided doses at 8-hour intervals. In the sulfonamide group, sulfathiazole, sulfadiazine, and sulfamerazine have each established a reputation for efficiency in the treatment of hemorrhagic septicemia. Sulfamerazine is believed to be especially effective, since relatively infrequent and small doses, one-half those of other sulfonamides, are required to maintain any given plasma concentration. In severe attacks sulfamerazine is combined with penicillin, 100,000 units administered intramuscularly at 3-hour intervals. Using sulfamerazine, McAuliff[18] has reported a recovery of 95 per cent of 122 cases of hemorrhagic septicemia and pneumonia in mature animals. He suggests an initial dose of 1½ grains (0.09 Gm.) per pound of body weight administered in equal parts at 8-hour intervals the first day, followed by ½ grain (0.03) Gm. per pound of body weight in two equal daily doses for two or three days thereafter. For more prompt action one may first give ⅓ grain (0.03 Gm.) sodium sulfamerazine intravenously, and follow immediately with the oral dosage.

According to Johnson and Farquharson,[19] Pasteurella organisms have been exceptional in cases of shipping fever in cattle in the Colorado section where diphtheroids, streptococci and staphylococci are more numerous. In the treatment of sick animals they report excellent results from the intravenous injection of the following preparation: potassium guaiacol sulfonate, 5 Gm.; ethyl alcohol 95 per cent, 10 Gm.; sodium iodide, 5 Gm.; sterile water 90 Gm. For calves weighing 300 pounds or less, 75 cc.; calves weighing 400 to 600 pounds, 125 cc.; larger cattle, 150 cc. When bronchopneumonia appears the results of the treatment are less encouraging.

In the treatment of pneumonia (shipping fever) in feeder cattle, Lies[20] gives 200,000 or more units of penicillin sodium in 500 to 1000 cc. normal saline solution per vein, and procaine penicillin, 1 million units intramuscularly. Also sodium sulfamerazine ½ to ¾ grain

per pound of body weight orally, or it may be administered intraperitoneally in a sterile normal saline solution. These treatments are repeated daily until the temperature is normal and the animal shows improvement, usually three or four days. For additional discussion on the treatment of pneumonia, see treatment of bronchopneumonia, page 46.

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PULMONARY EMPHYSEMA OF CATTLE

In the description of hemorrhagic septicemia in Cortland County, New York, in 1925, Moore and McAuliff[1] state that they "have not encountered a pneumonic condition in the pectoral form" in a total of

85 cases of which 33 were autopsied. The majority of these were the pectoral type, and the lesion was pulmonary emphysema.

Sporadic and enzootic forms of pulmonary emphysema are not infrequently met with in New York, especially in the fall. In affected herds there may or may not be a history of recent additions to account for the introduction of infection. Apparently it is contagious, for

Fig. 68.—Emphysema of the lung, as sometimes found in "hemorrhagic septicemia."

usually it is enzootic, and in the Cortland County outbreaks it presented all of the epidemiological features of typical hemorrhagic septicemia pneumonia in shipped cattle. The only difference was the presence of interstitial emphysema instead of consolidation of the lung in association with interstitial edema. This form of pulmonary emphysema is described here in connection with hemorrhagic septicemia because of its clinical resemblance to that disease. It is impossible to know, from

our present knowledge, whether the emphysema represents an independent disease, or whether it is a special lesion of hemorrhagic septicemia.

Etiology.—The question may well be raised with respect to pulmonary emphysema in cows: Is the emphysema a pathological lesion arising from the action upon the lungs of an infectious or toxic agent, or is it merely the mechanical effect of labored breathing? Extensive pulmonary emphysema is found in cows dead of a wide variety of diseases—lead poisoning, pneumonia, exhaustion, and death under experimental operations. It is found on autopsy whenever labored breathing precedes death, and occasionally it is found when the breathing was not recognized as especially difficult. Apparently there is a mechanical weakness in the bovine lung that predisposes to rupture of the tissues.

This febrile enzootic disease of cattle, leading to extreme emphysema of the lungs, often to death from suffocation in from 24 to 48 hours, appears to be a general infection, possibly hemorrhagic septicemia. The condition in the lung suggests a mechanical condition caused by dyspnea rather than an inflammatory lesion. While other general infectious diseases are marked by dyspnea and a high degree of emphysema, this seems to be the only one that leads to fatal suffocation.

In our experience, the only other condition leading to fatal suffocation from pulmonary emphysema has been a direct injury of the lung tissue from a foreign body in traumatic gastritis.

Another more frequent form of pulmonary emphysema of unknown cause follows the turning of cattle to lush pasture in the fall. This has been described by Schofield[2] in Canada where it is rare except in one or two counties in Ontario where kale and rape are grown extensively for fattening cattle. As described in Wyoming by Fuechsel,[3] it is increasing and in some localities is the greatest single cause of loss. In 1949, Fox and Roberts[4] reported that in the previous seven years 37 cases of acute pulmonary emphysema in cattle with a mortality of 46 per cent had been observed in the ambulatory clinic of the New York State Veterinary College. From Montana, Butler[5] reported in 1943 that not until seven years previously had it been seen in that State.

Symptoms.—Few descriptions present such uniformity as those which report the symptoms of acute pulmonary emphysema in pastured cattle. In the range country of the West it appears in the fall when cattle reach the green lush feed in the valley after removal from the mountain summer range, and occasionally it may attack a few summer fed cows in corrals. The majority of cases develop during the first week after arrival, affecting both male and female, and mostly at ages of

from 3 to 8 years. The characteristic symptoms are severe dyspnea and an expiratory grunt with the head extended and the tongue protruded (panting disease). There is complete anorexia and constipation or normal bowel movements. The pulse is fast and weak, the temperature normal, milk secretion ceases, the animal remains standing and is disinclined to move. Death may follow in from six hours to two days. After two to three days, subcutaneous emphysema may appear in the lower cervical or lumbar regions. Unless treated promptly the mortality is high.

Treatment.—The recommended treatments are atropine, adrenalin and pyribenzamine in combination with penicillin.[4] Or use twice the recommended dosage of antihistamines plus 10-15 cc. adrenalin solution every eight hours,[3] administered in the gluteal region with the animal in a chute to avoid excitement; only two of 48 failed to respond.

Prevention may be accomplished by feeding on hay alone for the first two or three days after coming from the range, and when turned on lush pasture leave only an hour the first day and increase the time gradually. In Canada the cattle on rape and kale are provided with hay and straw for which they have a craving.

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HEMORRHAGIC SEPTICEMIA OF SHEEP

(Pasteurellosis ovis)

Hemorrhagic septicemia of sheep is most prevalent in the Western Plains of the Mississippi Valley. Newsom[1] writes that the disease in sheep is a "real entity," and that "it is not a rare condition in his experience." As in cattle, it most often develops after the fatigue of a journey and frequently in the pneumonic form.

Symptoms.—The first to die may show nothing more than the symptoms of a general infection with extensive subserous and submucous hemorrhages on autopsy. In animals that survive longer the pneumonic form develops. All show dullness and have a discharge from the eyes

and nose. The intestinal symptoms are not marked. Often the temperature remains normal. With respect to the bacteriological diagnosis, Newsom states that observations extending over a period of years do not support the prevailing view that *P. oviseptica* can be regularly isolated from sheep dying of a variety of diseases.

Prophylaxis and Treatment.—The same principles apply to sheep as advised for cattle.

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HEMORRHAGIC SEPTICEMIA OF SWINE

(Swine Plague; Pasteurellosis suis)

Definition.—A sporadic or enzootic pneumonia of swine affected with a lowered resistance. It may be acute or chronic, is catarrhal in type, and often is characterized by necrotic foci in the lungs.

Etiology.—*Past. suiseptica* is the causative agent. Until the discovery of the filterable hog cholera virus, in 1905, swine plague was regarded as one of the destructive specific general diseases of swine. This discovery revealed that most of the so-called swine plague was hog cholera. At the present time few of the destructive enzootics in swine in the United States are attributed to *P. suiseptica*. In certain parts of Canada, where hog cholera is infrequent, enzootics of swine plague pneumonia are said to occur.

Morbid Anatomy.—Depending on the course, various stages of pneumonia are present. One finds hepatization of the apical and cardiac lobes, serous infiltration of the interlobular connective tissue, and foci of necrosis.

Symptoms.—The incubation period is from four days to a week. The onset is fairly sudden, marked by anorexia, "thumps," and a high fever. There may be a discharge from the eyes. Respiratory symptoms in the form of a cough and dyspnea develop early. After a few days a marked loss in condition occurs. The general course and prognosis is that of a catarrhal pneumonia, varying according to the sanitary surroundings, the virulence of the infection, and the age and resistance of the animal.

Diagnosis.—This consists in ascertaining the bacterial cause of the pneumonia. Aqueous suspensions of diseased tissue carrying *P. suiseptica* are fatal to rabbits in a few hours when injected subcutaneously. Smears made from the heart blood of the dead rabbit and stained with carbolfuchsin may reveal the bipolar organism. Fatal enzootics in swine

marked by hemorrhages in the serosa, pneumonia, degeneration and hemorrhage of the liver and spleen, and swelling and congestion of the lymph glands, indicate hog cholera rather than hemorrhagic septicemia. *Lungworm disease* may be mistaken for swine plague and other forms of pneumonia if one neglects to examine the contents of the bronchi.

Treatment.—Correct insanitary conditions. Swine plague secondary to other disease is properly regarded as a lesion of such disease and will disappear with the control of the primary affection. The use of vaccines or other biologics for the prevention or treatment of the usual case of swine plague is of doubtful value. Some ascribe a curative effect to aggressin when used in enzootics of the acute form, but this seems contrary to the known initial action of an aggressin or vaccine.

PURPURA HEMORRHAGICA

(Petechial Fever; Morbus Maculosus)

Definition.—Purpura is a noncontagious malady of equines occurring secondary to infections, such as strangles, influenza, pneumonia, and wound infection diseases. It is a disease of the circulatory system characterized by hemorrhage and edema in the skin, subcutis, muscles, mucosa and submucosa, and internal organs.

Etiology.—The essential cause is unknown. It is chiefly sporadic in the spring months. While usually it is secondary, it may develop in the absence of any recognizable primary disease. It is most prevalent in horses that have passed through stockyards, in remounts, and in similar groups where animals are constantly being added, and where the acute infections prevail. In its relation to wound infection diseases it is most apt to develop where the drainage of pus is poor and where necrosis is common. Thus it occurs in fistulous withers and in alveolar periostitis with emphysema of the sinuses. Like infectious diseases, it is cyclical in occurrence, appearing one year as a frequent and highly fatal disease and in another as an occasional mild affection. The presence of hemorrhagic edema has led to the assumption that a toxic chemical agent in the circulating blood reduces the elasticity of the vessels and renders them porous. The theory has also been advanced that purpura is of the nature of anaphylaxis, and this view is supported by experiments conducted by Marek. An extract of streptococci was repeatedly injected into a horse at short intervals and after an interval of a month another injection was given. Seven days later the horse came down with typical and fatal purpura. Kramer[1] has described a case that developed twenty days after a second injection of tetanus antitoxin; this was regarded as anaphylactic in origin. The theory that the immediate cause is a

destruction of the endothelial cells of the capillaries is supported by Flexnor's demonstration that snake venom contains a substance, hemorrhagin, which is highly destructive to the endothelial cells of the capillaries. According to Stevens[2] it seems probable that there may be other poisons capable of producing purpura and setting up degenerative lesions in the blood vessels. Stevens also states there is little room for doubt that diminution in the blood platelets is directly responsible for the hemorrhages occurring in primary pupura hemorrhagica in man. That the blood platelets are destroyed in equine purpura has been

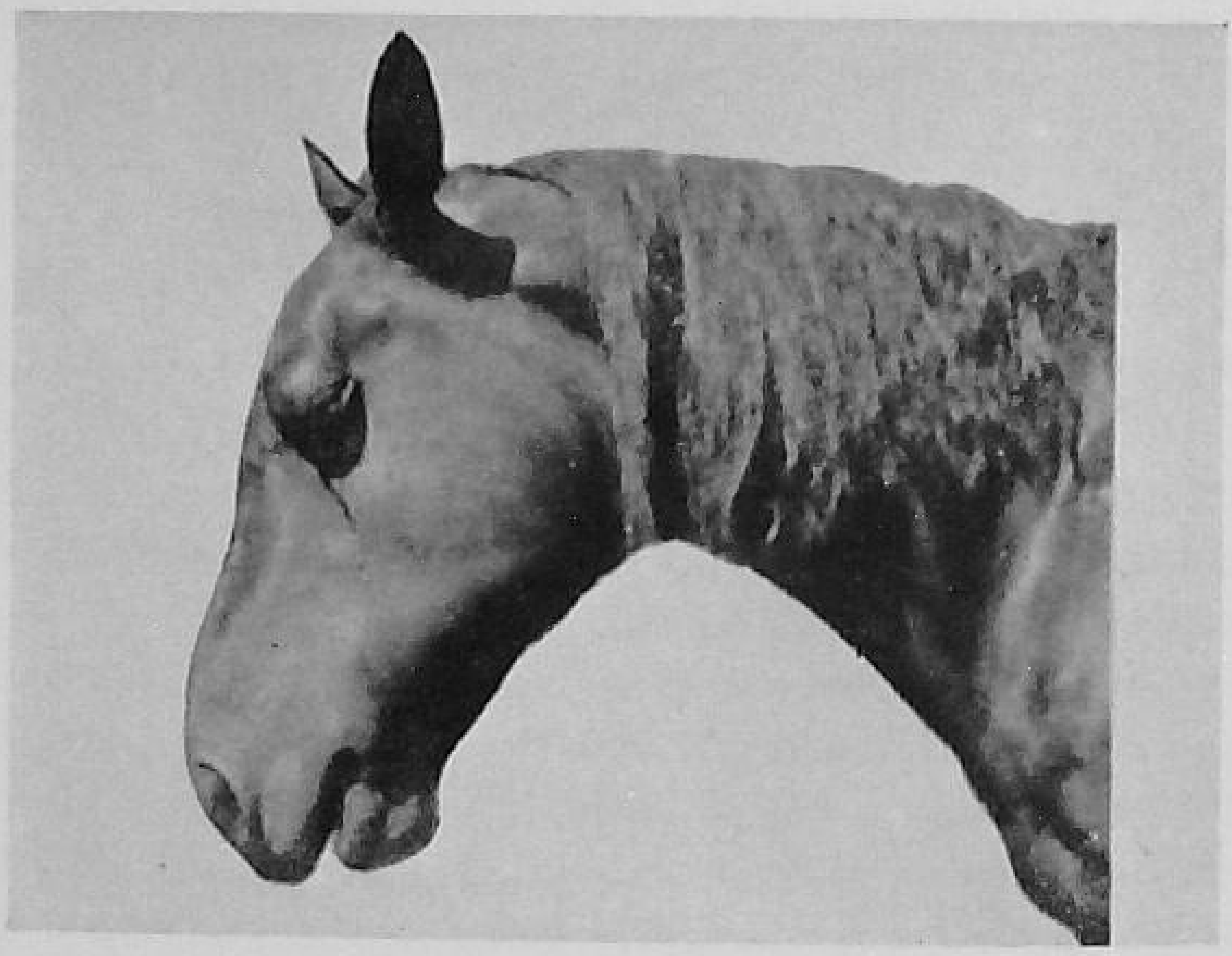

Fig. 69.—Purpura hemorrhagica.

shown by Wittmann and Contis.[3] While purpura is not dependent on any single cause, or on the activity of any particular kind of bacteria, it is most frequently associated with diseases in which streptococci are active.

Morbid Anatomy.—The cadaver presents diffuse swellings of the head, limbs, and ventral parts of the body, though in some cases these may have receded rapidly shortly before death. In the subcutaneous swellings one finds icterus and a hemorrhagic edema extending into the intermuscular tissue. The muscles may show hemorrhage, necrosis, or abscess formation. When the disease has been marked by intense swelling of the limbs there may be extensive sloughing of the tissues in the region of the joints. On opening the abdomen, subperitoneal hemorrhage is often found. Reddish serum or unclotted blood may be found in the large body cavities. The omentum and mesentery show icterus and

various degrees of hemorrhage. When the edema has localized in the intestines they are congested and thickened, the mucous surface is hemorrhagic, and there may be areas of necrosis. Agonal intussusception of the small intestine may be found. Hemorrhage is usually found in a greatly swollen spleen, and beneath the capsule of the kidneys and liver. In the chest cavity the chief changes are in the form of hemorrhages in the subpleural tissue, in the pericardium, and in the lungs. The lungs may also be edematous and contain foci of necrosis. In the upper respiratory tract are hemorrhagic edematous swellings of the mucosa of the pharynx, larynx, and nose.

Symptoms.—The disease begins with hemorrhages in the nasal mucosa. These are soon followed by edematous swellings on the nostrils, on the limbs, or along the ventral surface of the body. At first the swellings may be small and similar in appearance to simple stagnation edema of the subcutis of the limbs—"stocking." The swellings of the limbs soon present a distinctive, sharply defined abrupt upper margin, as if tied off alike in all four limbs. They are cold and painless and pit on pressure. These may develop gradually, changing little from one day to another, or they may become marked within twenty-four hours. In this event the limbs may be swollen to two or three times the normal size; the skin is tense and glistening and is firm to the touch; it may ooze serum, and after a few days areas of sloughing may appear in the region of the joints. Because of the swollen condition, walking is difficult. Rapidly developing swellings on the head lead to stenosis of the nasal passages or larynx, with severe inspiratory dyspnea, and finally to fatal edema of the lungs. In some cases, urticaria-like edematous swellings may be scattered over any part of the body, as the abdomen, the prepuce, the back, the neck, and the shoulders; this form of swelling is relatively infrequent. The eyes may be the seat of severe edema and hemorrhage. The lids close entirely, while the membrana nictitans and conjunctival sclera are edematous and hemorrhagic; when the lids are forced open, reddish fluid or blood escapes. There may also be hemorrhage in the oral mucosa, as well as in the nose and eyes, and occasionally the mucous membranes are icteric. Often there is a nasal discharge, and when the hemorrhage and necrosis are sufficiently marked this is abundant and fetid. Colic indicates involvement of the intestines with hemorrhage, edema, necrosis, and enteritis. The localization may shift suddenly from one part of the body to another, and this is an unfavorable sign. In one of my cases intense swelling of the limbs disappeared overnight; this was soon followed by colic and early death from enteritis with diffuse edema of the bowels.

The general symptoms correspond somewhat to the nature and

severity of the primary disease. In mild attacks the appetite and nutrition remain good. When swelling of the lips and nose are marked, eating is difficult and there is a loss of condition. The temperature is usually normal or only slightly raised; it may be high for a time and then drop to 102° or 103° F. The pulse, on the other hand, tends to be increased, from 50 to 80 or 100.

The blood picture has been described by Wittmann and Contis.[3] At the beginning there is a gradual progressive decrease of the red blood corpuscles (anemia, oligocythemia) induced by severe hemorrhagic edema of the skin and mucous membrane. The red cells may sink to 3 million, and there is a corresponding drop in the hemoglobin. Later in the course of the disease there are regenerative changes shown by an increase in the polychromatophils and the blood platelets. At the onset of the attack the leucocytes and neutrophiles increase; the number of leucocytes may be as high as 40,000 in comparison with a normal of 8000. The lymphocytes diminish in numbers and the eosinophiles disappear (aneosinophilia). When this degree of change is reached, deaths are frequent.

The average course is about two weeks, but it may be shorter or much longer. Improvement may be followed by a relapse, and a return of the symptoms is especially liable to occur when the animal is put to work without allowing a long period for convalescence.

The *diagnosis* is based on the presence of hemorrhages on the nasal mucosa in combination with edematous swellings of the skin. It may be confused with infectious diseases that show an external localization, such as strangles, anthrax, lymphangitis, influenza, and malignant edema. In these cases, the high temperature, immediate history, or nature of the primary disease are usually sufficient to exclude purpura. In purpura the swellings tend to be more symmetrical than those of other affections and their margins are more abrupt.

Progosis.—Because of the indefinite course and the variety of complications, the prognosis is always uncertain. The mortality is estimated at 40 to 50 per cent, but this estimate is probably too low. While a number of remedies have been prescribed for purpura, it remains to be proved that the course of the disease is favorably influenced by any of them. The following are unfavorable symptoms: rapidly developing and extensive swellings, necrosis of the skin or mucous membranes, severe inflammation of the larynx, pharynx or intestines, obstruction of the nasal openings, high fever, heart weakness, anorexia, albumen and blood in the urine, colic, pneumonia, and marked depression.

Treatment.—Direct blood *transfusion* is the most logical treatment for purpura. It supplies the deficiency in blood platelets on which

coagulation depends. Relatively large amounts should be given (2000 to 3000 cc. per vein) at intervals of one to two days. With a 50-cc. Luer type glass syringe, the transfer of 2000 cc. from the jugular of one horse to that of the patient is comparatively simple. When it is inconvenient to repeat injections, one may transfuse 4000 cc. at once. One may use citrated blood (4.5 grains of sodium citrate for each 100 cc. of blood). In the few cases of treatment of purpura with blood transfusion that have come under our observation, the results have been excellent. It has not been generally used. The special value of blood transfusion is thought to lie in the supply of blood platelets. Reports on the value of blood transfusion in the treatment of purpura are conflicting. In the treatment of purpura in horses in a remount depot, Seymour and Stevenson[4] found both blood transfusion and calcium gluconate unsatisfactory. They preferred formalin in doses of 10 cc. in 120 cc. distilled water given intravenously daily; in severe cases the patient received two doses the first day and one daily thereafter until recovery. Of 79 cases treated with formalin, 52 per cent recovered. Because of the apparently proven value of antistreptococcic serum, one would anticipate equal benefit from entire blood. Antihemorrhagic septicemia serum has also been recommended for the treatment of purpura in the horse.

Antistreptococcic serum is highly recommended by Fröhner[5] of Berlin, Jensen of Denmark, and others. They report a reduction in mortality of at least one third, from 40 to 50 per cent down to 15 per cent. It is given in the vein in doses of 200 to 250 cc. at intervals of one to two days; as much as 800 cc. has been given to a single horse. Fröhner[5] reports that the petechiae in the mucous membrane begin to recede in from twelve to twenty-four hours, the temperature falls, the leucocytosis is overcome, and the necrotic skin lesions begin to heal. Antistreptococcic serum is claimed to be especially indicated where the primary disease is caused by streptococci, as in strangles.

Other Remedies.—There are few diseases for which so many different remedies are alleged to have a curative action. Among the more recent of these are derivatives of *acridine*, such as acriflavine and rivanol. Acriflavine is administered in the vein (75 cc. of a 1:500 aqueous solution) every twenty-four hours. Wittmann[3] reports the use of rivanol on twenty cases of purpura without effect.

Gelatin has been administered in purpura because of its action in the control of internal hemorrhage. Schmidt[6] reported favorably on the treatment of 18 cases. A sterile solution (Merck) is given subcutaneously (200 cc.) with adrenalin 1:1000 (10 cc.) and symptomatic treatment. Its use has been limited, probably because of the high cost of the sterile preparation and the time required to prepare ordinary gelatin.

Fröhner writes that it was used on 11 cases in the Vienna clinic with no apparent effect.

As described by Rogers[10] in the Terminal Stockyards at Newport News, "Purpura hemorrhagica associated with equine influenza was treated by a daily subcutaneous injection of 10 cc. of adrenalin solution, 1:1000. This medication at times gave spectacular results, especially when the head was so swollen that the horse could not eat, and in some cases could scarcely breathe. Adrenalin reduced the swelling within 24 hours, and the petechiae in the nostrils disappeared as the swelling went down. We have also used blood transfusions, iodides and formalin, but with only an occasional recovery."

Calcium.—On the theory that purpura is a form of anaphylaxis, and that calcium has an antianaphylactic action, Laas[7] administered calcium in the treatment of purpura. According to his report 27 cases were treated in a period of two years with 26 recoveries. Calcium gluconate was given intravenously (150-200 cc. of a 7.5 per cent solution) daily in severe attacks and every second day in mild forms. A teaspoonful of Lugol's solution was given in the drinking water daily.

Formalin is recommended intravenously by some. Imrie[8] states that he has used it for twenty years in doses of 2 to 3 drams (8 to 12 cc.) in a 2 to 3 per cent aqueous solution per vein.

Potassium dichromate has been highly recommended by Steel.[9] It is given intravenously, 10 to 25 grains (0.65 to 1.62 Gm.) in 250 cc. of sterile water. Repeat in 48 hours.

General Treatment.—Provide a well-bedded box stall and remove the halter to prevent pressure upon the swollen tissues. Feed and water as usual and if swallowing is difficult give soft mashes or green food. If swelling of the head is increasing, and there is danger of severe stenosis, a tracheotomy tube should be applied. Medicinally, strychnine sulfate is a favorite with some. Circulatory weakness may be treated with caffeine and sodium benzoate (4 Gm.) subcutaneously. With the exception of stimulants, symptomatic treatment is seldom indicated. Purgatives are harmful. Avoid routine oral medication because of the possibility of causing foreign body pneumonia; either use an electuary or small amounts of a nonirritating solution.

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PURPURA HEMORRHAGICA IN OTHER ANIMALS

European literature contains numerous descriptions of purpura in cattle, and a few cases in swine have been reported. Apparently there is no record of the disease in England or the United States. Young cattle are chiefly affected. They show weakness, lack of appetite, suspended rumination, and hemorrhages in the skin, subcutis, conjunctival sclera, and the nasal and oral mucosa. Edematous swellings may form on the abdomen and limbs. There may be a high fever or a normal temperature. Recovery may be complete in one to three weeks and deaths are frequent. The postmortem changes are similar to those found in the horse.

We have had one case of apparent bovine purpura hemorrhagica in our ambulatory clinic. The animal was a young vigorous cow found sick in the pasture in the morning in the month of June. Bloody exudate was oozing from the skin and mucous membranes in many places. Within an hour after the call, about 9 A.M., the cow died. Autopsy showed extensive generalized hemorrhages throughout the body. The subcutis, muscles, and all of the internal organs were extensively hemorrhagic. Thorough examination of material taken to the laboratory was entirely negative.

LAMB DYSENTERY

Lamb dysentery is a highly fatal infectious disease of the newborn affecting lambs from one to six days of age. Clinically it is characterized by diarrhea, prostration and death in from a few hours to three or four days. The lesions are chiefly an acute enteritis of the small intestines. In the United States poor sanitation is considered to be the chief cause. In England it is regarded as a *Clostridium welchii* infection.

Etiology.—This disease causes heavy loss in England and Scotland where it has been gradually extending since 1920. From Montana

and other sheep-raising districts of the West, it has been reported since about 1927 and the losses are severe in affected herds. In 1930 it was diagnosed in the vicinity of Ithaca, New York. This malady is generally prevalent where sheep-raising is intensive, and apparently it is increasing in frequency and importance. Extensive reports on the disease in England have been made by Dalling and coworkers.[1,2,3,4] Welch[5] believes the disease in Montana is unlike the lamb dysentery in England.

Bacteriology.—In 1921 Gaiger and Dalling[1] reported that the disease is caused by a bacillus of the coli type which invades the body from the intestine. In 1923 they reported that the disease is caused by an anaerobe of *Cl. welchii* type and a bacillus of *B. coli* type acting conjointly, and in 1926 Dalling[3] was successful in reproducing the disease by the feeding of *Cl. welchii* alone, and by intravenous injection of a virulent *Cl. welchii* culture; death resulted in from 36 to 48 hours. From these experiments it is evident that in England *Cl. welchii* is the essential bacterial agent. It is believed that the ewe is the chief carrier, and that the soil becomes infected and can harbor the anaerobe for at least a year. Apparently lambs may ingest the organism from the surface of the ewe's teats, soiled either by vaginal or fecal discharges or contaminated by contact with the soil, or they may pick up infection direct from the soil or by actual contact with infected lambs. The disease may be reproduced by feeding material from the intestine of naturally infected lambs. It is not believed to be caused by navel infection.

According to Welch,[5] no specific organism has been found to be the cause of the disease in Montana. However, Tunnicliff[6] of Montana has since reported the finding of *Cl. welchii* as a constant inhabitant of the intestinal tube of normal young lambs and he[7] has also reported that a highly toxic strain of *Clostridium welchii* was the cause of a peracute unusual form of lamb dysentery in Montana. Marsh and Tunnicliff[8] were able to produce fatal dysentery by feeding cultures of several varieties of *Escherichia;* although many strains failed to produce dysentery, several strains from normal lambs were pathogenic. They concluded that "under favorable conditions certain strains of the bacteria, principally *Escherichia*, which are normally present in the intestinal tract, may produce the disease." In the few cases examined at Ithaca, *B. coli* was found.

Infected Ewes.—The disease is introduced into clean flocks by the purchase of infected ewes, the lambs of such ewes being the first to sicken. Transmission from such ewes has been accomplished by feeding feces, but not by feeding milk. It has also been observed that lambs invariably contract the disease when they nurse a ewe whose own lamb died from dysentery. Intra-uterine infection has not been proved,

though many shepherds believe that lambs may be born with the disease.

Housing.—According to the reports from England, the first appearance of the disease on a farm is only in pens, and toward the end of the lambing season. In subsequent years it appears two weeks after the beginning of the lambing season, and in ewes kept in the open. Welch[5] states that apparently it is a shed infection of Montana, and that it is rare in lambs born on the grass in late April and May. He believes cold wet weather is the principal factor and that shed sanitation is next in importance. Handling the lambs and allowing them to overeat are other predisposing causes. Marsh and Tunnicliff[8] have concluded that "dysentery of newborn lambs, as it occurs in the northwestern United States, is the resultant of several factors rather than a specific disease caused by a specific pathogenic micro-organism. Those factors are: low temperatures, resulting in lowered vitality, or resistance, of the lambs; insanitary conditions of lambing sheds and corrals, resulting in the ingestion during the first day of the life of the lamb of a relatively large number of bacteria in filth; and the presence in the environment of strains of intestinal bacteria which are potentially pathogenic." They believe that this condition differs clinically and pathologically from the English lamb dysentery, and that the bacteriology is also different.

Morbid Anatomy.—As in calf scours, the lesions may be slight or marked. The peritoneal fluid may be normal or turbid. The kidneys, liver, and spleen are either normal or light in color. The intestine may show general congestion in very acute cases. As described in Great Britain, usually both the large and small bowel show necrotic areas ¼ inch in diameter with typical hemorrhagic zones around them; these may be seen from the peritoneal surface. The adjacent lymph glands are enlarged and edematous. The thoracic organs are normal. In the lambs autopsied near Ithaca there were extensive hemorrhages in the lungs, the mucosa of the stomach, and beneath the capsule of the liver. Hemorrhagic enteritis was extensive. In general, the postmortem changes are identical in appearance with those observed in calf septicemia.

Symptoms.—When the disease first appears it usually affects lambs less than 48 hours old, but in subsequent years lambs up to two weeks of age may be attacked. In England it is reported as more prevalent towards the end of the lambing season, probably because the ewes are brought into the paddocks. In the United States it is less prevalent at the end of the lambing season when lambs are born on grass. At the onset of the outbreak affected lambs may die quickly without showing

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any dysentery; they are found dead in the morning. In less acute types there is a period of dullness, and diarrhea shortly before death, the feces being brownish and blood-tinged. On farms where the disease has become established the course is somewhat longer, two to three days. Few recover.

Treatment.—In an outbreak of lamb dysentery in a flock of sheep near Ithaca every newborn lamb had died within 36 hours after birth, regardless of numerous sanitary precautions. Cultures from autopsied lambs yielded a colon organism. In an attempt to follow the principles employed in the prevention of white scours in calves, the following feeding program was adopted with complete success: the newborn lamb remained with the dam for twelve hours; it was then fasted for eight to twelve hours; it then received the white of an egg every three hours for three feedings; this was followed every three hours with the white of an egg and a half ounce of the dam's milk for two feedings; the lamb was then returned to the dam. Sulfathalidine is useful.

Control.—Shaw and Muth[9] have reported excellent results from the use of acidophilus milk in the prevention and treatment of lamb dysentery. The lambs were given one ounce before being allowed to nurse the ewe, and if scours developed two-ounce doses were given until the lamb either recovered or died. Emphasis is placed upon prompt destruction of every case of lamb dysentery, especially the earlier ones, since healthy lambs may become infected from ingestion of the intestinal contents. Acriflavine (0.5 grain) administered in capsule at from one to seventeen hours after birth materially reduced the loss from lamb dysentery, as observed by Marsh and Tunnicliff;[6] a second dose may be given about eight hours later.

Vaccination.—Dalling and coworkers[4] have described a successful method of intra-uterine immunization against dysentery in lambs. For this purpose they used a toxin-antitoxin prepared from *Cl. welchii.* One dose was injected into the ewe in October or November before the ewes were pregnant, and a further dose in March or April about a fortnight before lambing was due to begin in the flock. Equally good results were obtained from a vaccine obtained from *Cl. welchii* ("toxoided whole culture"). They also report that "antitoxin of the lamb dysentery anaerobe prevents lamb dysentery in at least 99 per cent of cases." This should be given before the lamb is 12 hours old.

Lamb dysentery may be prevented by removal of the flock to fields and sheds not previously occupied by sheep. It may be necessary to abandon customary sheds and yards for a year, during which time the paddocks may be plowed and the sheds disinfected and kept open as much as possible to the sunlight.

It is recommended that lambing sheds be kept clean and dry; that the udders and thighs of the ewe be clipped and kept clean; and that a separate attendant be provided for sick animals. Anti-calf-scour serum has been reported to be useful in dysentery in both lambs and kids.

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TRANSMISSIBLE GASTROENTERITIS IN PIGS

Definition.—In 1946 Doyle and Hutchings[1] applied the term transmissible gastroenteritis to a highly fatal diarrhea of baby pigs which they found in Indiana to be due to a filterable virus. In 1952 Bay[4] wrote that "although the disease has been reported as a community disease in some swine-producing areas, it was not until the spring farrowing season of 1951 that it was observed as such in Indiana."

Etiology.—In the 1940's there were numerous articles dealing with baby-pig disease or three-day pig disease which was variously attributed to hypoglycemia, acute uremia, alimentary toxemia, faulty nutrition, and reverse anaphylactic shock. These reports indicate that scours in newborn pigs is frequent and that it is due to more than one cause. While Doyle and associates do not regard "transmissible gastroenteritis" as the most important cause of baby pig mortality, apparently it is the only common and increasingly prevalent type for which a specific cause has been identified. In individual herds the losses have been as high as 90-100 per cent of the affected litters; it may appear in only one or in successive litters of the same sow, and the susceptibility is rapidly decreased with age. In some herds it has spread to feeder hogs, to sows yet to farrow, and in a mild form it has also been observed in

shoats when there were no baby pigs on the farm. During the acute part of an outbreak all pigs which are born may die.

Transmissible gastroenteritis was produced experimentally[1,2] by contact exposure to the sick, by feeding ground gastrointestinal tissues of the sick, and by feeding Berkefeld N filtrates from tissues and gastrointestinal contents to pigs 1 to 5 days old. Following exposure to filtrates the incubation period was as short as 18 hours, and the frozen virus remained viable for 70-80 days.

Morbid Anatomy.—The chief postmortem changes are gastritis, enteritis, atonic intestines, and a very fluid whitish, yellowish, or greenish intestinal content. The kidneys are degenerated and may or may not present gross urates. Gross evidence of inflammation of the stomach and intestines is not always found.

Symptoms.—The pigs are dehydrated, lose condition rapidly, and may vomit. Diarrhea in the form of a fluid, whitish or yellowish-green fetid feces is a predominant symptom. The disease usually terminates fatally in from three days to a week. In sows there may be diarrhea, vomiting, inappetence, and agalactia with recovery in three to ten days.

Control.—As reported by Bay,[4] on a farm where 40-60 sows farrowed each month 400 pigs were lost in March regardless of separate caretakers for each case of spread, disinfection of farrowing houses, and individual houses with separate caretakers. Finally, all sows due to farrow in April were sold, the farrowing quarters were cleaned and vacated, and the May and June litters escaped infection. In small herds where control of the personnel is possible, isolation of the sick animals has proved useful. It is usually advisable to scatter the noninfected litters and the sows to farrow over as large an area as possible, in order to minimize spread from litter to litter, if any one litter becomes sick. If a large number of sows are yet to farrow after the infection begins, the sale of enough animals to give a four- to eight-week break in the farrowing appears to be the most effective way of stopping losses; this is especially true in herds that farrow the year round.

No effective treatment has been found.

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PIG SCOURS

Pig scours is a colibacillosis with gastroenteritis. Although dysentery in pigs is the cause of many deaths, the literature on the subject is brief. Kinsley[1] writes that scours is one of the most widespread of the fatal diseases of suckling pigs, the estimated losses being 7 to 10 million pigs annually in the United States. It is most prevalent in the hog-raising areas of the Middle West, and on farms where many sows are kept under poor sanitary conditions, but it is not infrequent where the sanitation is good. Kernkamp[2] reports that it is more common in the winter and early spring than in pigs farrowed in the summer and fall. He also believes that climatic influences are more important than diet; sudden changes from warm to cool have given a high correlation with the onset of the disease. Chilling of the body surface apparently sets up functional changes in the alimentary canal; an onset of scours has been observed in 75 per cent of the newborn within 7 to 10 hours after a sudden change in temperature.

Hurt[3] expresses the opinion that threeday-disease in pigs that die at an age of from 3 to 5 days is due to a dietary disturbance of the sow, since the symptoms indicate a definite toxemia in the milk. His attempts to transmit the disease from one pig to another by contact, by parenteral inoculation, and by feeding tests in which stomach and intestinal contents were used, were negative.

In 1948 Whitehair *et al.*[4] reported an outbreak of pig scours in a herd in Wisconsin which was transmitted by feeding triturated viscera. With the exception of successful treatment with sulfathiazole the description closely resembles that of transmissible gastroenteritis; filtrate transmission was not reported, and no infection was found.

In a report by McBryde[5] on acute enteritis in pigs on a garbage-feeding ranch in California, he concludes that "from the bacteriological and histological studies, it appeared that the disease was the result of invasion with *Bact. coli*, which had assumed pathogenic and invasive properties. . . . In every case in which enteritis had developed, cultures showed invasion with *Bact. coli*. The organism was recovered quite consistently in cultures taken from the mesenteric glands, kidney, spleen and heart-blood, indicating the *Bact. coli* had not only invaded the small intestine but the body as well." Variations in the extent, distribution and character of the infection are observed on examination of pigs from different sources and in different pigs from the same herd, but in general the colon organism predominates. As in the case of scours

in the young of other species, doubt has been expressed that *Bact. coli* is the primary infection in pig scours. But until other evidence is available, this type of infection cannot be ignored.

On postmortem examination, the cadaver is usually emaciated, the hind parts are smeared with feces, and the intestinal contents are fluid and fetid. There is a severe enteritis of the small intestines, and various degrees of pleuritis, pneumonia, and peritonitis may be present.

In the *treatment* of scours in pigs the sulfonamides and antibiotics have displaced all previous remedies. Sulfathalidine, 1 grain per lb. body weight daily is especially beneficial (Graham).[6] In the outbreak described by Whitehair *et al.*,[4] sulfathiazole 1 Gm. per 10 lb. body weight, was clearly beneficial, while sulfaguanidine was less effective and penicillin was useless.

Control.—In garbage fed stock in California the condition of pigs is greatly improved, by supplying supplemental feeds to the sows and keeping them in pasture before farrowing; those kept in farrow houses or lots for five days or more almost always lose their litters with baby-pig disease.[7] Avoid abrupt changes in feed and overfeeding immediately before and after farrowing. Provide dry and warm pens, preferably with electric heated pig brooders. The use of antibiotics as a preventive against scours in pigs has drawn several favorable reports, as from Cunha[8] (1950) who observed that aureomycin prevented scours in pigs, and O'Rourke[9] (1952) who reported the value of bacitracin pellets implanted subcutaneously at farrowing time or shortly thereafter.

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SCOURS IN FOALS

Of the diseases of newborn foals, the most frequent is pyosepticemia. But in many individuals a colon septicemia or enteritis prevails, similar to that met with in calves. Diarrhea in foals is not associated with pyo-

septicemia. The *etiology* seems to be similar to that of white scours in calves. A foal may develop diarrhea from the eating of feces, straw, grass, and other foreign material. Since scours often appears in foals at the age of 9 days, various explanations as to the cause have been made. According to one of these, the removal of the dam for service on the ninth day results in irregular suckling, engorgement of the udder with milk, and consequent overeating. Williams[1] has suggested that diarrhea in foals arises from ingestion of genital discharges that reach the udder by means of gravitation down the thighs, this discharge being especially abundant at the time of the first estrum on the ninth day.

The *symptoms* occur chiefly in the second week, in the form of a transient diarrhea from which the foal usually recovers. A few die of severe gastroenteritis.

Treatment consists in the administration of castor oil followed by bismuth subnitrate 3 drams (12 Gm.) thrice daily. The use of acidophilus milk, or of the carminative mixture prescribed for calves (page 172), is indicated. In severe cases daily administration of the dam's blood (500 cc.) subcutaneously or intravenously is beneficial. Overeating may be prevented by milking the mare between nursing periods. Where there is evidence of eating foreign substances, a muzzle should be applied. Sulfathalidine is indicated.

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NAVEL-ILL IN FOALS

(Joint-Ill; Pyosepticemia; Omphalophlebitis)

Definition.—Pyosepticemia of the newborn is a septicemia characterized by localization of purulent foci in the joints, tendon-sheaths, kidneys, and other parts of the body; in certain types of infection the disease is chiefly a septicemia without localization. The etiology and lesions are variable.

Etiology.—There are two main views concerning the chief method of infection in navel-ill in foals: (*a*) Primary infection of the umbilicus after birth, and (*b*) intra-uterine infection. M'Fadyean and associates,[1] maintain that the great majority are caused by postnatal infection by streptococci and other organisms that are facultative parasites frequently present in the soil, infection being favored by lack of cleanliness and disinfection of the parturition stalls. That the disease is caused by intra-uterine infection is held by Williams,[2] Fincher,[3] Dimock,[4] and others of wide experience in the horse-breeding

industry. This view is held because of failure to prevent the disease by means of ideal stable hygiene at the time of parturition, and because foals are often sick at the time of birth. Furthermore, it has been brought under control by exclusion from service of stallions and mares showing evidence of infection when presented for breeding. It has also been observed that an aborted fetus may reveal a pure culture of streptococci not distinguishable from those causing navel-ill on the same farm. According to Fincher's[3] observation, "less than one third of the mares whose foals were seriously diseased or died became pregnant following that parturition. This fact, as well as the history of the individual cases, suggests that mares with an infected uterus are the ones where the foals will be diseased." Nearly all admit the possibility of infection before birth.

Bacteriology.—Various kinds of organisms cause the characteristic lesions. Chief among these are *streptococci*, and *Bact. viscosum equi (Bact. nephritidis equi; Bact. equirulis)*. *Bact. coli* is frequently reported as a cause of navel-ill, but apparently its chief effect is to produce a rapidly fatal general sepsis during the first four days. *Bact. abortivo equinus* has been reported by Good and Smith[5] as an occasional cause of acute sepsis and joint-ill.

Each one of these organisms may produce symptoms and lesions peculiar to the infection. Mixed infections, such as *Bact. coli* and streptococci, are not infrequent, while the bacteria of different foals from the same dam may vary from one year to another. Of the various causes, streptococci are the most frequent, and they usually induce a subacute or chronic form of joint-ill in older foals—two to three weeks of age. Viscosum infection attacks the foals during the first few hours after birth, often the animal is sick when born, and it is characterized by typical necrosis or abscess formation in the kidneys. In colon infection the disease also appears early, causing death within the first four or five days. While *Bact. viscosum equi* has been recognized in Europe as a cause of navel-ill in foals for at least twenty-five years, it was first described in this country by Snyder[6] in 1925. In 1928 Dimock[4] stated that "at present disease due to this organism has reached alarming proportions." It is not pathogenic to small experimental animals. Dimock[7] (1935) reports that it has been isolated from approximately 40 per cent of all foals presented for autopsy and bacteriological examination at the University of Kentucky. Its habitat is believed to be in the digestive tract of the pregnant mare; it is readily recovered from the tonsillar crypts of the great majority of horses of all ages both male and female. It is believed to invade the fetus *in utero* through the blood of the dam.

In a report from Dimock *et al.*[8] on the type of infection recovered from 550 of 810 dead foals, 25.8 per cent yielded streptococci; 21.48, *Shigella equirulis;* 3.95, *Escherichia coli;* 2.22, *Corynebacterium equi;* 0.99, *Salmonella typhimurium;* 0.74, *S. abortivo-equinus;* and 0.19, equine virus abortion; in 32.1 per cent no infection was found. On the type of infection recovered from 520 of 1074 aborted fetuses, 26.81 per cent yielded equine virus abortion; 17.03, streptococci; 2.04, *S. abortivo-equinus;* 0.93, *Escherichia coli;* 0.74, staphylococci; 0.37, *Shigella equirulis;* and 0.18, *Corynebacterium equi;* in 51.5 per cent no infection was found.

In the chronic fatal cases reported from Denmark which included 50 per cent of the whole material, a streptococcus infection was usually present. Two strains were identified: Type A corresponding to Schutz's streptococcus found in equine pneumonia, and Type B resembling strangles streptococci (Magnusson).[9]

Morbid Anatomy.—In general the lesions are characteristic of the type of infection, though exceptions are numerous.

In *streptococcic infection,* where the course tends to be chronic, pyemic changes are marked. One or more of the joints and tendon sheaths contain pus, the stifle and hock joints being favorite seats of localization. Usually there is an inflammation of the umbilicus with abscess formation that extends to the umbilical veins or to the urachus, and often involves the peritoneum. Abscesses may be found in the spleen, liver, or lungs.

In *viscosum infection* the umbilicus and navel vessels are not usually involved. In 73 cases observed by Magnusson, purulent omphalophlebitis was observed in only 6. The stifle and hock joints are often swollen, yet general sepsis without localization is common. The appearance of the contents of the joints and tendon-sheaths varies widely; usually they contain a grayish yellow mucopurulent exudate. There is no necrosis of the bone or cartilage. The lungs are usually normal, pleuritis and pericarditis are rare, while the intestines may show marked congestion. The most distinctive changes are in the *kidneys.* The glomeruli are enlarged and many are converted into abscesses. Dimock reports that of 10 foals dying within 24 to 48 hours, only 1 showed necrosis of the kidney; while of 7 foals dying at 72 to 96 hours, all but 1 showed necrosis of the kidney.

The specific organism may usually be recovered in pure culture from the kidneys, all joints, and other organs. It is rarely possible to recover it from the mare's uterus, though it has been observed that mares with foals infected with *Shigella equirulis* may develop metritis.

The meager available reports on the lesions of *colon infection* in foals

lead one to infer that its chief effect is an acute septicemia with possible localization in the digestive system, as in white scours in calves. In Magnusson's report on 236 autopsies, it was held to be the cause of death in 27.1 per cent. In the protocols prepared by Fitch[10] four cases of unmixed colon infection are described: joint infection is mentioned once, and all other lesions mentioned are general hemorrhage, peritonitis, pericarditis, and enteritis.

Symptoms.—While each of the three chief infections of navel-ill may cause any clinical form of the disease, certain distinctive clinical and anatomical characteristics frequently indicate the nature of the causative organism.

Dimock[4] classifies the symptoms of foals infected with *Bact. viscosum* as follows:

"1. Those dead at birth or showing characteristic symptoms at birth but in a semicomotose condition—the type commonly called 'sleeper.'

"2. Those showing symptoms of sickness at birth but more or less active.

"3. Those apparently normal at birth but developing the disease six weeks to five months later."

Infection due to *Bacterium viscosum equi* "is characterized by sudden onset, extreme prostration, short duration after appearance of symptoms of disease, and death."

With few exceptions the attack occurs within the first three days of life. In Magnusson's[9] series, 56 of 73 cases fell on the first day, and in 29 the foal was weak at birth. More than half died when two days old showing symptoms of septicemia. Only nine were more than a week old when they developed typical polyarthritis, tendovaginitis, and intermuscular abscess. The temperature is high, 103° to 105° F., and the breathing is fast. Localization in the joints appears after the first twenty-four hours.

In *streptococcic infection* there is a much longer period of incubation. The disease commonly appears after the foal is 10 to 14 days of age and the fatal cases die after a course of two to three weeks. Often the first symptom to appear is swelling of one of the joints, particularly the stifle or hock. This may be followed by weakness and gradual loss of condition until the foal dies of exhaustion. Frequent urination is a common symptom in all foals suffering from navel-ill. Pervious urachus and purulent omphalitis are frequent.

In *colon infection* the symptoms are those of an acute septicemia, sometimes in association with inflammation of the joints and umbilicus. The mortality is high.

Prognosis.—In acute forms, occuring a few hours after birth, the

mortality is high—90 to 100 per cent. In the chronic streptococcic forms, showing navel infection or lameness, but no general symptoms, recovery is occasional. The average death rate has been estimated at from 30 to 75 per cent, but these estimates are based on superficial observations and they have little relation to the expectation in any given outbreak.

The *diagnosis* of pyosepticemia or omphalophlebitis is not difficult. The swollen joints, tendon-sheaths, and navel are pathognomonic. But in the absence of such localizations a diagnosis by the symptoms alone may be difficult. Those who have had wide clinical and laboratory experience with the diseases of foals (Magnusson, Dimock) speak of the frequency of weakness at birth from no apparent cause. Many of these animals recover. Magnusson reports that many are cases of septicemia from which the causative organism may be isolated. Yet in his series of 314 autopsies of foals, there remained 7.3 per cent in which he was not able to make any other diagnosis than lack of energy. Scours in foals should not be confused with pyosepticemia, though individual cases may occasionally present similar symptoms.

Prophylaxis and Treatment.—The use of antistreptococcic serum and other biologics is reported by M'Fadyean[11] as ineffective. Reports from many farms indicated a mortality of 50 per cent both before and after their administration. Fitch[10] also reports that serums or bacterins have little if any value as prophylactic or therapeutic agents.

The situation is complicated by the variety of bacterial causes, and the variable circumstances under which breeding is conducted. In many cases it is impossible to know which organism is responsible for the disease until after the foal is dead. In localities where a certain type of infection is found to be usually present the appropriate prophylaxis and treatment may be more effectively applied. In all cases, subcutaneous injection of the dam's blood into the foal has gained considerable reputation; this is given in doses of 100 to 200 cc., and increasing amounts are administered if the foal becomes sick. Sulfanilamide (5 grams for each 100 lbs. body weight daily) is said to be useful.

In *streptococcic* infection, Sohnle[12] advises the use of the dam's blood (50-60 cc.) in combination with stable-specific antistreptococcic serum. For the same purpose Gmelin[13] advises the use of the dam's blood and a stable-specific vaccine. Fincher writes,[3] "On all cases, I used equine antistreptococcic serum as a preventive against septicemia and joint-ill, and no foal that died after this treatment showed pure culture of streptococcus."

In studs where the prevalent infection is *Bact. viscosum equi*, a corresponding serum is claimed by Heinrich[14] and others to be highly effec-

tive. The dam receives serum at six to eight months of pregnancy, and the foal receives 30 cc. at birth.

For protection against *Bact. abortivo equinus*, a specific vaccination of the dam has proved to be effective.

Bact. coli infection may be controlled with calf scour serum, as in calves. Restriction of the diet and of the use of muzzles are also beneficial.

Since postnatal infection through the navel is known to occur, and since it may be the chief portal of entry on many farms, systematic care of the newborn foal should be practiced. For delivery, mares should be placed in special disinfected foaling stalls. Directly after birth the navel is thoroughly saturated with tincture of iodine. In the Government Stud of Austria as soon as the foal is born the navel is compressed with gauze saturated with 5 per cent formalin-alcohol solution until pulsation ceases (Mally).[15] It is then twisted or torn apart a few centimeters from the abdomen and the stump saturated with 10 per cent tincture of iodine. When able to half stand the foal is held to suckle. As soon as the placenta comes away the hind parts and udder of the mare are washed with warm water and she is removed with the foal to another box stall.

Among the most effective measures for control of navel-ill and other diseases of newborn foals, are the modern methods of examination and treatment of the mare and stallion previous to breeding.

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NAVEL-ILL IN OTHER SPECIES

Navel-ill in *calves* is most frequent in large herds where it occurs more or less intermittently in a sporadic form. As in foals, it is most abundant where abortion and other breeding diseases are ignored. It is probable that the great majority are caused by postnatal infection. It is prevented by thorough saturation of the navel with tincture of iodine.

Navel-ill in *lambs* sometimes assumes importance in large breeding establishments. Welch[1] refers to many deaths from this disease in which the infective agent was *Actinomyces necrophorus;* the lesions were necrobacillosis of the liver. In lambs from one to three weeks old pyemic arthritis, as well as other metastatic lesions are frequently caused by navel infection. There are two chief clinical forms: an acute type leading to death when the lamb is about a week old, and a more chronic form, affecting older lambs, characterized by lameness, stiffness, or paralysis. Posterior paralysis may be caused by pressure of a pyemic abscess on the cord. The disease is readily controlled by thorough disinfection of the navel at birth. The lambing pens should be clean and heavily bedded. Marsh[2] directs that the cord should be cut, leaving a stump of about 1.5 inches, and then dip the stump in tincture of iodine contained in a small wide-mouthed bottle. In the same article he describes a specific arthritis in lambs, caused by the bacillus of swine erysipelas, in which the avenues of infection are the umbilicus or castration or docking wounds.

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SALMONELLOSIS

Salmonella infections in both man and animal have been the subject of extensive investigation and of rather voluminous literature in Europe. A few endemics have been described in the United States.

The Salmonella group includes *S. choleraesuis (suipestifer)*, *S. enteritidis*, and S. *typhimurium (aertycke)*.

Salmonella choleraesuis (suipestifer), the cause of infectious necrotic enteritis in swine, is described on p. 185. While it is possible that diseases caused by *S. enteritidis* and *S. typhimurium* may prove to be important in animals in this country, as in Europe, only a few descriptions of them have been published in American literature. Schofield[1] suggests that lack of such reports is due to neglect of bacterial study of enteric infections in cattle.

PARATYPHOID IN CATTLE

(Infectious Enteritis)

In 1916 Meyer, Traum, and Roadhouse[2] described a fatal endemic of infectious diarrhea in calves, caused by *S. enteritidis*. The age occurrence is at from 2 to 4 weeks and older, in contrast to white scours, which appears chiefly in the first week of life. One should not attach too much importance to the age, however, since any endemic disease of the young may become atypical during the course of a protracted outbreak. The cases described by Schofield[1] were in pregnant heifers and mature cows.

In 1902 Mohler and Buckley[3] reported an enzootic among adult cattle. Brain symptoms and convulsions, similar to those observed in some cases in calves, were present. The causative organism was described as a bacillus of the enteritidis group.

The habitat of the infection is widespread. It is acquired by ingestion, and is generally believed to enter the stable in carriers or in the milk. Jensen and others believe it is a normal inhabitant of the digestive tract, while others have failed to find it in healthy calves. Meyer[2] states that "the isolated bacteria are pathogenic for guinea pigs and rabbits. They produce exceedingly active toxins. Even the smallest doses when repeatedly applied to rabbits will cause loss in weight and a predisposition to a secondary infection to which the animals invariably succumb." Feeding of broth cultures to calves was followed in twenty-four hours by high fever and diarrhea.

As in white scours, so-called predisposing causes have a marked influence. Thus in the cases described by Meyer,[2] calves at ten days of age

received a diet of hay and were changed from whole milk to skim milk at the rate of a pound daily. The first four deaths were in the group undergoing this change.

Morbid Anatomy.—Fibrinous or hemorrhagic enteritis is constantly present. According to Fröhner and Zwick[4] these changes are most marked in the abomasum and small intestine; the spleen is enlarged to two or three times its normal size, and this is said to be the most characteristic lesion; usually the liver is enlarged and contains multiple necrotic foci, and these may also be present in the kidneys and the spleen; the carpal and stifle joints may be enlarged, and there may be inflammatory changes in the lungs. Writing from "our experience," Jones[5] states that "the lesions are those of hemorrhagic or fibrinous enteritis, particularly in the most posterior region of the ileum, the cecum and upper portion of the small intestine; the causative organism is readily isolated from the feces, affected mucosa, and mesenteric lymph nodes." Schofield[1] mentions acute inflammation of the mucous membrane of the stomach, small intestines, cecum and colon, quantities of serofibrinous exudate mixed with blood and feces, a few fibrinous casts, and an extensive and unusually severe enteritis; the regional lymph nodes were swollen and some were hemorrhagic; the kidneys and lungs were congested; numerous small abscesses in the submucosa of the colon, similar to those found in chronic salmonellosis in swine, yielded almost pure cultures of *S. typhimurium,* and this organism was also recovered from the serofibrinous exudate in the intestines, the spleen, liver, and heart's blood.

Symptoms.—The period of incubation is from two to eight days. The disease may be mild or highly fatal. The onset is marked by weakness, sometimes lachrymation, recumbency, and high fever. Diarrhea is marked, though evacuations may be suppressed in the early stages. The feces are thin, brownish yellow, fetid, and often mixed with blood. Dyspnea and cough are sometimes present. The stifle and carpal joints may be swollen. Brain symptoms in the form of involuntary movements, running into objects, and convulsions have been described. Peracute attacks cause death in a few hours. More often the course is from one to two weeks with a rapid loss of flesh, and occasionally the course is over a period of three to four weeks.

Control.—Prophylaxis and treatment should be followed according to the principles described under white scours. A prophylactic bacterin and sulfathalidine were employed by Schofield.[1]

PARATYPHOID IN SHEEP AND FOALS

In 1932, Dr. Charles E. Hagyard[6] described an acute infectious dis-

ease of foals characterized by diarrhea, inflammation and necrosis of the mucous membrane of the large intestine. Examinations of tissues at the Kentucky Experiment Station revealed the cause to be *Salmonella typhimurium*. The lesions were chiefly located in the cecum and great colon in the form of an enteritis with necrosis and erosion. The intestinal wall was greatly thickened. In some cases there were peritonitis, and inflammation of the small intestines. The distinctive symptoms were a fetid diarrhea and high fever. In addition there were marked congestion of the mucous membranes, anorexia, thirst, irregular peristalsis, restlessness and prostration. The course in fatal cases was from four to six days and accompanied by severe and persistent straining. The disease was thought to have been checked by the use of an autogenous bacterin.

Newsom and Cross[7] have described attacks of dysentery in lambs, which they attribute to *S. typhimurium*. The lesions were chiefly confined to an acute inflammation of the fourth stomach and small intestines. The stomach and intestinal contents were frequently stained red or black, due to escape of blood from the walls of the organs.

The symptoms were dullness, drooped ears, anorexia, and a thin, watery, fetid diarrhea. The temperature at first was as high as 105.6° F. The course was from a day to two or three weeks and the mortality was variable. Fasting was shown to be an important predisposing cause.

Reports of human infection caused by *S. enteritidis* are not rare in the United States. According to Jordan,[8] a number of food poisoning outbreaks have been plausibly attributed to *S. typhimurium*. Alvarez[9] writes that the commonest cause of food poisoning due to bacteria or bacterial toxins is contamination of the food with living bacteria belonging to the *Salmonella* group. "The commonest offenders are *Bacillus enteritidis* and *B. aertrycke*. Occasionally *B. suipestifer* is at fault."

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STRANGLES

(Distemper; Infectious Adenitis)

Definition.—Strangles is an acute febrile infectious disease of solipeds characterized by inflammation of the nasal mucosa and abscess formation in the adjacent lymph glands. It is caused by *Streptococcus equi.*

Etiology.—Strangles prevails especially among young animals, being an infection to which nearly all are exposed early in life. Often it is foremost among the acute infections in army camps, remounts, stockyards, and on breeding farms. In the early part of World War I it was reported as the most common disease of horses in the German Army. As a sporadic disease among farm horses it is less serious than where many are assembled together. The most susceptible age is in colts over 6 months and young horses from 2 to 5 years old; young foals and old animals are less frequently attacked. Strangles has been met with in the newborn. The disease is acquired naturally through the ingestion of contaminated food or water. Primary strangles of the mesenteric glands in foals is attributed to infection through the intestinal mucosa acquired by sucking an infected udder. Apparently the infection is carried to the udder of the mare on the nose of the foal.

While strangles may occur at any time of the year it is most prevalent in the spring. Improper housing and cold drafty stables exert an unfavorable influence. Recovered animals are said to be permanently immune, and there is little evidence of its recurrence in adult animals kept under usual conditions. In remounts, however, where young susceptible individuals are constantly being added, the infection attains a virulence that results in repeated attacks. Richters[1] reports that recurrence may be observed after three months and again after six months, though usually in a milder form.

Streptococcus equi.—This gram-positive organism is found in long chains in the inflammatory exudate of the nasal mucosa and in the pus from abscesses; usually it is associated with other bacteria. There are numerous reports on the independent character of *Str. equi* as distinguished from other streptococcic infections in the horse. Miessner[2] believes the strangles streptococcus is an independent form sharply defined from others by its action on carbohydrates. Fröhner and Zwick[3] write that it is differentiated from *Str. pyogenes* by the form and length of the chain, as well as by its failure to ferment lactose. Zlatogoroff[4] found that *Str. pyogenes equi* does not cause strangles, nor immunity against strangles, and that the disease is caused only by *Str. equi*. Ogura[5] concludes that in many respects *Str. equi* is different from other pus-

forming streptococci in the horse, but particularly in morphology, action on sugars, and pathogenicity. He considers it the most frequent cause of acute inflammations of various organs: nose, throat, lungs, umbilicus and joints of foals, as well as the udder and genitals in mares. Edwards[6] found that while streptococci in pure culture were obtained from strangles abscesses, they were not all of the same strain.

Subcutaneous inoculation of mice with cultures causes suppuration of the subcutis and adjacent lymph glands and metastatic abscesses. Rabbits are less susceptible, while guinea pigs react only slightly.

According to Richters[1] it is one of the most resistant of the vegetative organisms when enclosed in pus or blood. In water it survives for 6 to 9 days; on utensils, skin, and roughage it lives 3 to 4 weeks; in room temperature, 5 to 6 months; in packed dung, 14 days. It has been obtained in a fully virulent form from the nasal mucosa of old horses and from animals in apparent health.

The disease has been produced experimentally in the horse by bringing pus in contact with the mucosa of the nose and throat; by inhalation of infected dust; by the application of pus to the scarified skin, and by swallowing infected drinking water. It can be produced by inoculation with pure cultures of *Str. equi*. The disease is highly contagious. The streptococci may be transmitted to the respiratory mucosa through the air, or they may reach the intestinal mucosa in the food or water. Infection may enter through skin wounds, through the udder, or through the vagina from coitus.

Morbid Anatomy.—The postmortem changes are widely variable. When the chief localization is multiple abscess formation of the superficial lymph glands many external swellings are visible. The mesenteric or mediastinal lymph glands may be the seat of abscesses several inches in diameter. Abscesses may cause death from involvement of the spinal cord or brain, causing meningitis. In one of our cases, an abscess formed in the muscles of the lumbar region and penetrated to the spinal cord. Inhalation of pus from a ruptured retropharyngeal lymph gland may give the postmortem changes of bronchopneumonia. Any of the internal organs may be the seat of single or multiple abscess formation. Lesions of septicemia and pyosepticemia are usually present.

Symptoms—The period of incubation is from 4 to 8 days; under the influence of exposure it may be less. The onset is sudden in the form of depression and anorexia and a temperature of 104° to 106° F. There is a medium increase in the frequency of the pulse, 40 to 50. If pyemic or septic conditions develop it goes much higher. Localization on the nasal mucosa appears on the first or second day. The mucous membranes are congested and there is a serous nasal discharge; after about three days

this becomes mucopurulent or purulent. It is also abundant, so that the feed and manger are soon in a filthy condition. In individual cases the nasal discharge may be slight or absent. Because of involvement of the pharynx, there is a cough that may be easily induced. Along with the nasal discharge a swelling of the submaxillary lymph glands appears; this may be hot and painful and surrounded by a zone of inflammatory edema. After a few days an abscess develops and there are one or more fluctuating spots, or the skin over the swelling oozes a yellowish serum, the hair falls out, and the pus is released through a necrotic slough in the skin. The pus is yellow and creamy in consistency. There may be a multiple abscess formation. After evacuation of the abscess the swelling recedes rapidly and the temperature drops to normal. In uncomplicated cases the course is from two to four weeks. In mild outbreaks certain individuals may have only a nasal catarrh without abscess formation. In the average attack there is no marked disturbance of the general condition.

Atypical Forms.—Because of the metastatic nature of strangles, many different complications are possible. The following are among the more frequent of the irregular forms, or "bastard strangles":

1. *The deep lymph glands.* Pharyngitis may be the primary localization on the mucosa, or it may spread to the pharynx from the nose. The symptoms are difficult swallowing, regurgitation, and swelling of the *retropharyngeal lymph glands.* The swellings are located above the larynx in the region of the parotid glands. The abscess usually ruptures into the guttural pouch, the pus escaping through the mouth and nose. In this form the course is more irregular and there may be a marked disturbance of the general condition. It is sometimes associated with abscess formation of the *subparotid lymph glands;* the entire parotid region may be swollen from the presence of multiple abscesses. In one of our cases the *masseter muscles* contained many purulent foci that caused the death of the animal. The *guttural pouches* are sometimes the seat of suppuration and abscess; localization here may result in empyema of the pouches, or necrosis of the internal carotid with fatal hemorrhage. Sudden escape of pus into the pharynx may cause gangrenous pneumonia; usually the abscesses rupture externally.

According to Wirth,[7] in at least 90 per cent of pharyngitis there are abscesses in the subparotid and retropharyngeal lymph glands, and especially in those which may be designated as lymph nodes of the guttural pouches. These guttural pouch lymph nodes lie above the arytenoid cartilages between the mucosa of the guttural pouches and the posterior pharyngeal wall. Smears made from the pus of these abscesses are identical in appearance with those obtained from a strangles abscess,

which leads Wirth to the conclusion that pharyngitis in the horse is nearly always strangles.

2. The *superficial lymphatics* of the face may be the seat of a localization attacking the eyes, lips, nose, cheeks, and leading to a diffuse suppurative inflammation of the subcutis of the head and neck.

3. The *pharynx, larynx, trachea, and bronchi* are sometimes involved in an acute catarrh with or without abscess formation in the adjacent lymph glands.

4. In some cases there is a tendency to metastatic localization in *distant organs and glands*, such as the cervical, axillary, inguinal and other superficial lymph glands. More serious and frequent are abscesses in the mediastinal, or mesenteric glands. Such abscesses may attain great size and rupture, causing death within a few hours; they may remain dormant for months and then rupture; or they may cause extensive intestinal adhesions. When convalescence is unsatisfactory a rectal examination may reveal an enormous enlargement of the mesenteric lymph glands, or of the lymph glands around the pelvis or in the sublumbar region. Other localizations are the meninges of the brain or spinal cord; the joints, tendon-sheaths, and deep muscles, and the liver, spleen or kidneys.

5. *Septicemia and septicopyemia* are the most frequent causes of death. During convalescence from severe attacks purpura hemorrhagica is not infrequent. Coital infection has been observed. Nodules and abscesses appear in the walls of the vulva and vagina a few days after service.

The *prognosis* is favorable when the abscesses are limited to the submaxillary or a few of the superficial glands.

The course is somewhat longer when the retropharyngeal group is involved, but recovery may be anticipated unless multiple abscesses form throughout all the adjacent tisses. Localization within the body cavities is always serious. On the whole the mortality is low, from 0.5 to 2 per cent. Among foals, however, irregular forms are frequent and the mortality may be high. It is also more serious in horses in cold, damp, or drafty stables. Unusually severe outbreaks with a high mortality have been reported. The prognosis is less favorable in recently shipped horses when the disease may be associated with influenza or contagious equine pneumonia.

Treatment.—A mild sporadic case requires no special treatment. Provide complete rest, a slightly limited diet, and in cold seasons protect against cold drafts that may enter through open windows or doors. In the treatment of individual cases keep a pail of fresh water before the horse; to each fresh pail a half ounce of chlorate of potash may be

added. The manger and sides of the stall should be cleaned frequently of any pus that may be dropped from the nasal discharges. Two or three times a day, wash the purulent secretions from the eyes and nose with a mild antiseptic solution, leaving the skin dry.

As a rule no special treatment of the abscesses is required until they are sufficiently advanced to be opened. If external treatment seems advisable, apply a hot antiseptic compress to the throat. Any form of blister is liable to do more harm than good, except for thickenings that may remain after recovery from the acute symptoms. The development of the abscesses and the attendant inflammatory edema may cause inspiratory dyspnea from stenosis of the pharynx. In some cases this may be relieved promptly by means of steam inhalations. In stenosis, abscesses should be given surgical drainage as soon as there is any evidence of an accumulation of pus, but when no definite abscess is apparent the effect of steam inhalations should first be tried. Following convalescence a subparotid abscess may remain as a cause of inspiratory dyspnea when the animal is worked; this requires surgical drainage. When penetrating the subcutaneous tissues, one needs to use a blunt instrument or the finger to avoid accidental separation of large blood vessels that may have been pressed out of their natural position; this is particularly true of a deep subparotid or retropharyngeal abscess. A tracheotomy tube may be required. For the nasal catarrh expectorants of ammonium chloride, as prescribed for coryza, are useful. If the cough is aggravating add an ounce of fluid extract of belladonna to each pint of syrup. Steck[8] has reported beneficial action from sulfanilamide, 80 Gm. every 48 hours administered in 2 liters of water through a nasal catheter; this was usually repeated two or three times. Improved forms of sulfa preparations, such as sulfadiazine, or sulfamerazine are preferable to sulfanilamide. The dosage is 1 grain per pound of body weight the first day, followed by one half this amount daily for not more than five days. Hyperimmune strangles serum in doses of from 100 to 200 cc. intravenously, repeated daily depending on the patient's symptoms and progress, is recommended by Seymour and Stevenson.[9] They used approximately 100,000 cc. in the treatment of horses affected with strangles in the remount depot. This was supplemented with calcium gluconate, 200 cc. of a 23 per cent solution every third or fourth day in debility, and with sulfanilamide in doses of 60 to 90 Gm. daily for 4 consecutive days in pyosepticemia. Benefit from penicillin and sulfamerazine has been reported by Roberts.[10]

Immunization.—Because of the tendency to permanent immunity in recovered animals, numerous attempts have been made to establish artificial immunity. In Europe sera prepared from *Str. equi* have been

used for prophylactic and curative purposes, and the reports of the results are conflicting. At best the serum protects for two or three weeks and its possible curative action is obtained only when administered early, before the abscesses form. Vaccines have been widely used in Europe and other countries with apparent benefit. Edwards[6] has reported on the use of vaccine and serum in an attempt to check a 5 per cent loss in young animals admitted to a remount in India; he believes the mortality was reduced somewhat. In a subsequent report Allen[11] gives additional evidence of the value of pyovaccination and paddock dispersion. The vaccine was prepared as follows: The abscess to be punctured was treated with tincture of iodine. Four cc. of pus was obtained and transferred to a sterile flask containing beads; 10 cc. of sulfuric ether (Merck's) was gradually added and continually agitated. The material was allowed to stand for twelve hours with frequent shaking. Five cc. of boiled and cooled normal saline solution was added and an almost homogeneous mass was thus obtained. The vaccine thus prepared was kept in a stoppered bottle until required for use. The mortality of affected animals was reduced to 2 per cent.

Richters[1] has described a method of control employed in the German Army for which distinct benefit is claimed. The sera and vaccines in general use were apparently of no value. These were replaced by a combination of polyvalent immune sera and polyvalent methylene-blue vaccine prepared in the veterinary research laboratory of the army. The curative effect was apparently favorable. In the treatment of 1000 cases over a period of two years the mortality dropped 50 per cent, while metastases and sequelae were scarcely observed.

Bazeley[12] has reported active immunization against *Str. equi* with heat-killed vaccines consisting mainly of capsulated organisms.

In the prevention of strangles, all experienced observers of the disease in its virulent form emphasize the need of early segregation of the sick, and of strenuous efforts to prevent its spread. This implies early diagnosis. Avoid the shifting of animals during an outbreak. Destroy purulent exudates promptly by means of frequent washing and disinfection of the stall and parts of the horse that may be contaminated. Provide individual water pails, or streams of running water. Provide a separate personnel for the sick. With the onset of the disease administer polyvalent serum and polyvalent vaccine. *Streptococcus equi* bacterin may immunize foals when given at 5-day intervals in doses of 2 cc., 3 cc., and 5 cc.

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TETANUS

Definition.—Tetanus is chiefly a wound infection disease caused by *Bacillus tetani (Clostridium tetani)* and characterized by tonic spasms of the muscles with marked exacerbations. The bacillus of tetanus was discovered by Nicolaier in 1885 and isolated in pure culture by Kitastato in 1889. It is found in the superficial layers of the soil, in manure, in street dirt, in the feces of horses and other herbivorous animals, and not infrequently in the feces of man.

Etiology.—In the United States it is most frequent in the South, where precautions against tetanus are an essential part of the routine treatment of wounds and wound infection diseases. It is frequent in all parts of the United States and has a world-wide distribution. Equines are most susceptible; swine are not infrequently infected, especially pigs; and occasionally cows may suffer from parturient infection. It is not rare in sheep and goats.

The disease usually follows a slight injury, particularly of the feet (nail pricks) and limbs. Penetrating injuries from nails and splinters are most dangerous, but even slight abrasions from farm implements often infect. Horses frequently show no external injury. Pigs are most readily infected through the umbilicus (navel infection) or through castration wounds; cattle may be infected from a nose ring, parturition

injuries, or dehorning; lambs are infected by navel-ill, docking, and castration wounds. Occasionally tetanus occurs without showing any recognizable wound, so-called idiopathic tetanus; but even in these cases it is believed that some wound exists. Often the wound heals entirely before the onset of the disease.

The tetanus bacillus has contaminated vaccines, catgut, and gelatin, and has followed the use of the hypodermic needle. It most readily occurs in badly infected wounds.

The *tetanus bacillus* is a spore-forming anaerobe capable of living indefinitely in the soil and in the intestines; the spores are the most resistant known. Cultures have remained viable on steel nibs for nearly ten years, and spores may remain in the tissues for months. The toxin is recognized as the most virulent of known poisons; it is more than a hundred times as toxic as strychnine. According to Gessler[1] the older theory of the ability of tetanus toxin to travel up nerve sheaths has given away to the theory of blood stream circulation of the toxin. As described by Firor,[2] "The bacillus remains localized at or near the point of inoculation where it liberates a toxin which acts on the neuromuscular end-organs of the adjacent skeletal muscles. Some of the toxic substance passes into the lymphatics and blood stream, from which a part is taken up by the cells in the cord and medulla. Here it is probably altered to form a secondary substance that is not neutralized by antitetanic serum. This secondary substance circulates in the blood stream and seems to cause death by interference with some essential part of the respiratory mechanism. Once a lethal part of this hypothetical toxin has been formed, no known treatment will save the life of the animal."

Morbid Anatomy.—There are no characteristic lesions. In the lungs one may find secondary edema, pneumonia, or gangrene.

Symptoms.—The period of incubation is from one to three weeks and it may be as long as four months. The onset is gradual in the form of stiffness, which may be general or local. Localized stiffness occurs most often in the masseter muscles or in those of the hind limbs; only exceptionally do the symptoms first appear in the muscles in the region of the wound. After about twenty-four hours the syndrome is usually distinctive.

The expression is anxious, but the consciousness is normal. The reflexes are increased and the patient readily becomes excited or frightened. In one of our cases, in a cow, there was a tendency to attack strangers. At the beginning the pulse and temperature are normal; towards the end of a fatal attack the temperature may reach a height not approached in any other disease, reaching as high as 110° F., and continuing to rise for several hours after death. In mild attacks the

pulse and temperature remain normal through the course. The peristalsis is diminished and the bowels are constipated.

In the *horse* tonic spasms of the skeletal muscles are extensive. Beginning first at the head or in the muscles of the hind limbs they extend either slowly or rapidly until the condition becomes generalized. Occasionally the spasms are limited to a definite group of muscles—local tetanus. Spasms of the muscles of the jaw cause difficulty in prehension and mastication of food (lockjaw) and there may be drooling of saliva. Involvement of other groups of head muscles causes a peculiar erect position of the ears, narrowing of the eyelids, prolapse of the third eyelid, dilatation of the nostrils, and, sometimes, difficult swallowing. Often the horse is unable to eat off the floor. Spasms of the muscles of the neck, back, and tail cause a stiff and extended position of the head

Fig. 70.—Tetanus.

and neck (orthotonus), and an elevation of the tail; occasionally the head and neck may be drawn backward (opisthotonus), and the tail may be curved laterally. Turning is difficult. Stiffness of the extensor muscles of the limbs causes difficulty in walking, and a sawbuck-like attitude; in some cases the legs are spread apart. Stiffness, or difficulty in backing, may be one of the first signs of tetanus. Sweating, and trembling of the affected muscles are frequent.

General spasms, or those affecting the respiratory muscles, may derange the respiration and circulation causing superficial fast breathing, congestion of the mucosae, and towards the end a fast pounding heart. Contraction of the abdominal muscles gives a tucked-up appearance.

In *cattle*, we have met with the disease following puerperal infection, dehorning, and placing a ring in the nose. In one bull the disease appeared in about a week after the operation. The symptoms were very distinctive in the form of extension of the head and neck, exposure of the mebrana nictatans, tucked-up abdomen, stiff extended tail, and a sawbuck position of the limbs; the spasms were general. There may be trembling of the muscles and the animals are easily startled by sudden movements or a sharp noise which adds to the degree of prolapse of the third eyelid.

In *sheep* and *goats* opisthotonus is the rule, and bloating is frequently

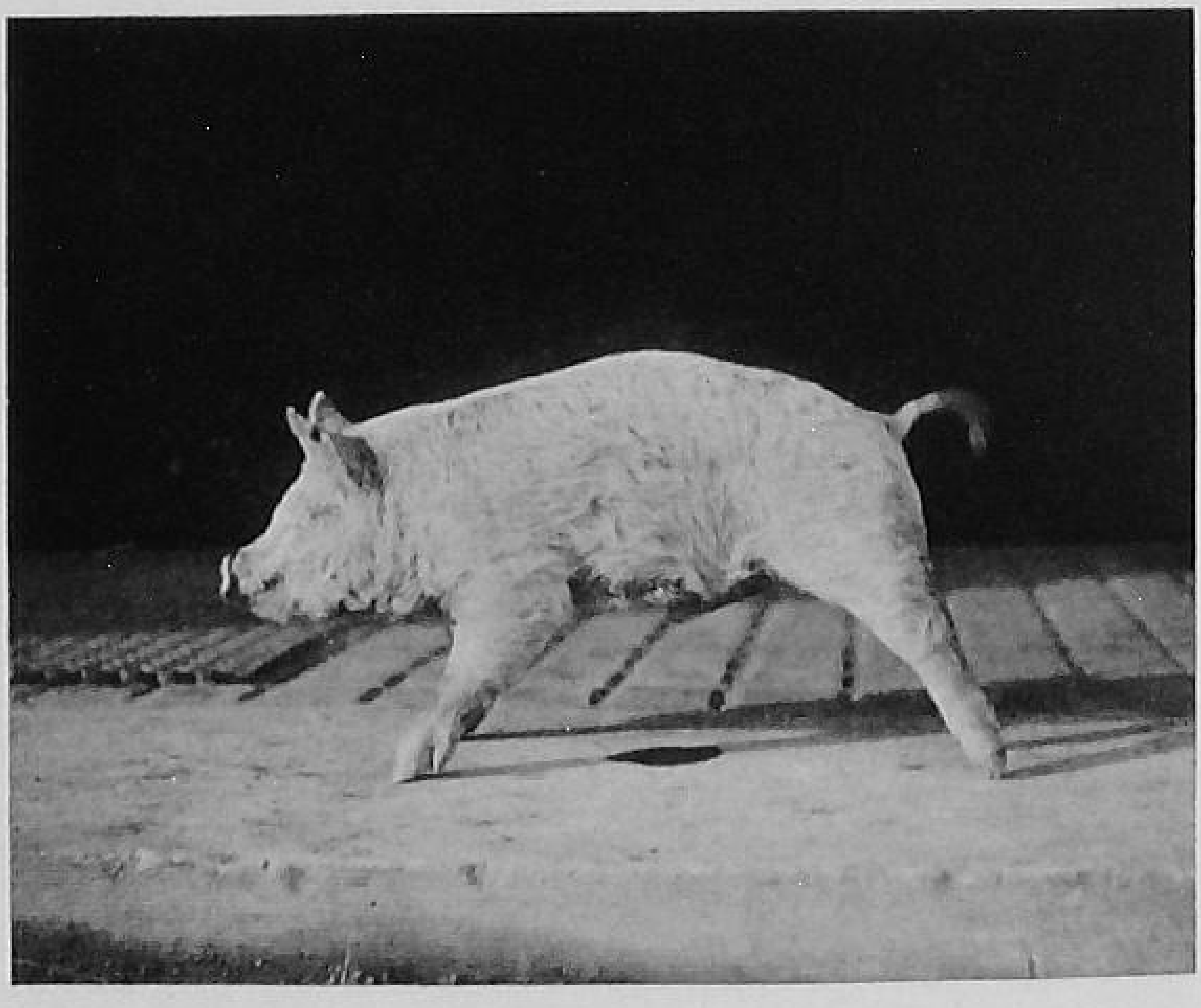

Fig. 71.—Tetanus.

present. The disease may be endemic from navel infection in lambs. At first they are dull and stiff; then the head is drawn backward or to one side and in a short time they are unable to stand. A sudden disturbance or noise may cause spasms. It may be associated with profuse diarrhea.

Swine are chiefly affected with generalized tetanus.

The *prognosis* depends entirely on the intensity of the attack, and it is probable that this is determined by the amount of toxin absorbed. In an intense generalized attack the disease is fatal in from two or three days (peracute) to a week or ten days (acute). If the animal survives a mild attack for a period of two weeks, the prognosis is favorable; these cases are said to be chronic. The prognosis is also favorable in partial tetanus and if the incubation period is long. Ability to take food and

water, and absence of intense generalized spasms are favorable signs. The prognosis is always uncertain, for some cases develop recurrent intensity of spasms after several days of apparent improvement. Three to four weeks are required for recovery, and spasms may persist for as long as six weeks. The mortality is estimated at from 75 to 80 per cent. In our ambulatory clinic it has been 60 per cent over a fifteen-year period; in cattle, sheep and swine it has been 100 per cent. The death rate varies in different parts of the country and in different years.

Treatment.—The use of tetanus antitoxin, even in extremely high dosage, has not affected the mortality. Fröhner and Zwick[3] report that in the German Army in World War I, 245 cases of tetanus in the horse

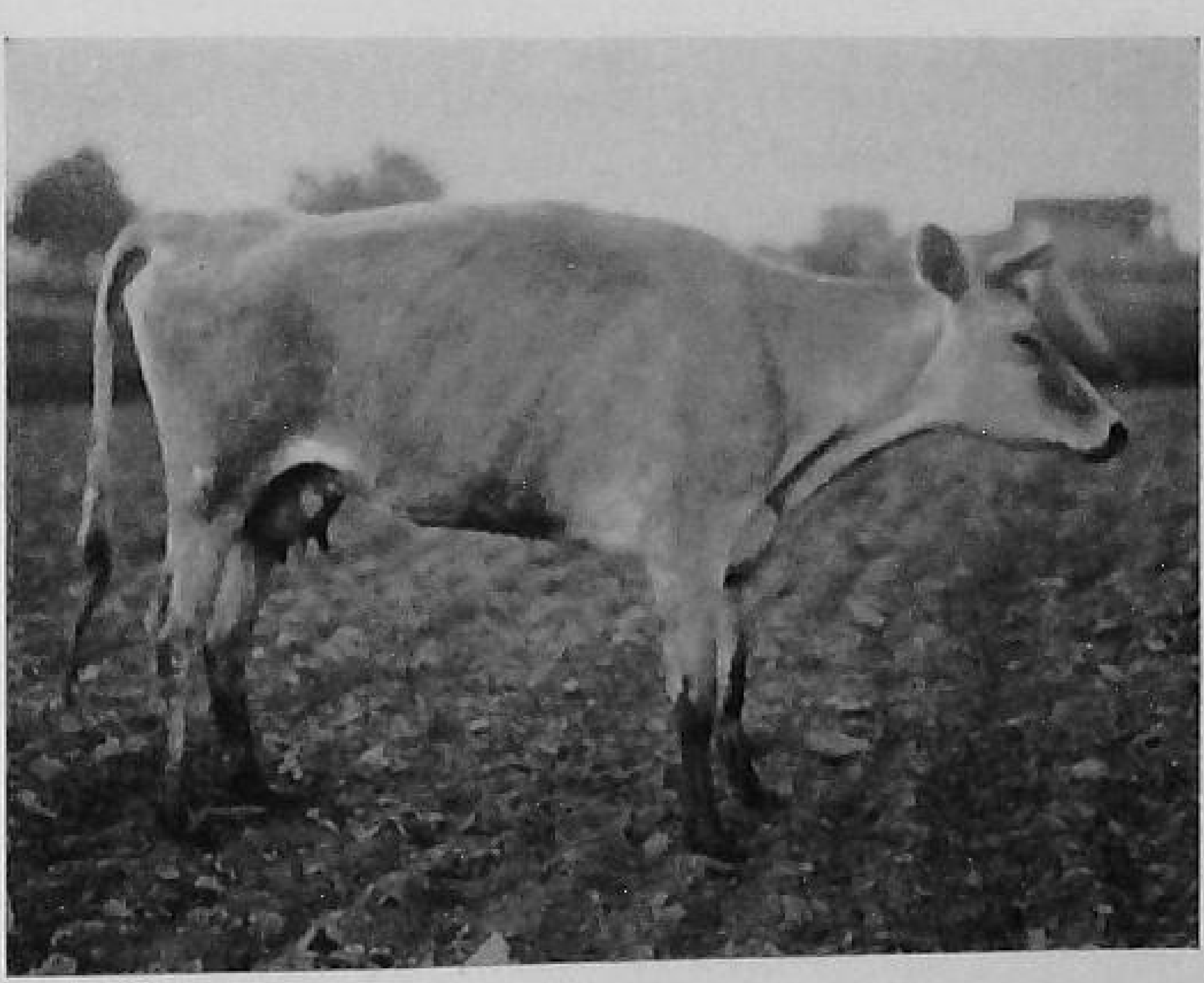

Fig. 72.—Tetanus.

were treated with large doses of serum. The mortality was 62.5 per cent; among untreated cases the mortality was 65 per cent. The curative effect of serum was regarded as worthless. In our ambulatory clinic we frequently administer 100,000 to 200,000 units intravenously in the treatment of tetanus, but there is nothing to indicate that this changes the average mortality of 60 per cent. It has been reported, however, that an initial dose of 30,000 to 50,000 units at the onset, repeated in twelve to twenty-four hours, will reduce the mortality to about 10 per cent. It seems possible that such favorable results may be possible in northern climates where tetanus is relatively infrequent and mild. Antitoxin is usually administered intravenously, but some prefer, in addition, to inject it into the muscles and into the spinal canal.

The only effect of antitoxin is to neutralize the toxin in the body

which is free and unaltered before it has been fixed in the central nervous system, and this may be accomplished by injection of 150,000 to 200,000 units the first day in large animals, followed by 25,000 units daily for about ten days. To provide rest from spasms or convulsions, depressants of the nervous system such as chloral, are indicated. Burns[4] has reported favorably on the use of procaine hydrochloride, 100-150 cc. of a 2½ per cent solution intravenously once daily for the first two days, then every other day for two or three doses.

The course is more favorable when the animal is kept in a box stall, somewhat darkened and quiet. Feed and water as usual at a height from the floor that will not require lowering the head. Slings are useful when there is difficulty in standing, or rising. Tetanus antitoxin is effective as a prophylactic when given at the time of injury; the dose is 500 to 1500 units during the first seventy-two hours after infection; this confers a passive immunity which lasts about ten days.

Active immunization against tetanus has been practiced by Ramon.[5] He gave tetanus *anatoxin* to 30,000 horses, repeating the dose at the end of a month. One year later the blood of these horses contained sufficient antitoxin for natural protection. Active immunization with toxoid has also been described by Glenny, Hamp, and Stevens,[6] and this method of protection against tetanus is now generally regarded as highly effective.

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SWINE ERYSIPELAS

Definition.—Swine erysipelas is an acute, subacute or chronic infectious disease caused by the erysipelas bacillus (*Erysipelothrix rhusiopathiae suis*). Pathologically it is characterized by gastroenteritis, swelling of the spleen, nephritis, and degeneration of the liver, heart and muscles. Its chief occurrence is in swine, but man, lambs, pigeons, turkeys, rabbits, and mice are susceptible.

Etiology.—The bacillus was discovered and an efficient vaccine was

developed by Pasteur and Tuillier in 1882-83. As early as 1879 Koch had discovered the mouse septicemia bacillus (*B. murisepticus*) which is now classified as identical with that of swine erysipelas. Smith,[1] in 1885, and Moore,[2] in 1892, recovered organisms from fresh tissue of swine which they identified as those of mouse septicemia, but which they concluded were not those of swine erysipelas. After nearly a third of a century, in the early 1920's, there were again reports of isolation of the organism from various types of lesions: by TenBroeck[3] from the tonsils of pigs affected with hog cholera; by Creech[4] from the lesions of diamond skin disease; by Ward[5] from polyarthritis in swine; and by Giltner[6] from cases of acute septicemia. And a decade later, in the early 1930's, the acute septicemic type which had devastated European herds for years, was now reported from many widely separated areas in the United States (Taylor,[7] Munce,[8] Baker[9]). In 1933 the disease was recognized by Baker in New York, where there had been many deaths in pigs following vaccination against hog cholera. In 1938 Breed[10] reported that the infection had been identified as a cause of swine maladies in 28 of the 48 states. The brief period covered by these identifications, 1931 to 1937, indicates that unrecognized infection may have been widespread. The extent of such infection is suggested by the 1942 report upon the disease in Nebraska by Van Es and McGrath[11] who state that "in connection with this project 281 outbreaks of acute swine disease were investigated and in not less than 24 per cent of this number swine erysipelas proved to be the malady involved." In 1938 it was estimated by Breed[10] that swine erysipelas constitutes from 10 to 17 per cent of the acute infectious diseases of swine in the middle western section of the United States. In 1940 an examination by the Bureau of Animal Industry of 472 joints from swine showing varying degrees of arthritis, the swine erysipelas organism was recovered from more than 75 per cent of all specimens received, and the highest incidence of infection was from specimens in the Corn Belt. In 1944 the disease was reported as still on the increase in the Middle West and gradually spreading to other sections. Thus a comment by Moore on the high significance of the mouse septicemia bacillus in fresh tissue of swine has been confirmed.

The epidemiology is widely variable. On farms not previously infected it may resemble hog cholera in the extent and rapidity of the attack, or it may affect only two or three individuals and then apparently disappear. This initial attack may be followed later by unthriftiness and lameness that proves to be due to swine erysipelas infection. To judge from the available records of laboratory examinations the disease shows a cyclic increase and recession over a period of four or five years. Because of the inconstant and insidious character of this infection it may

be introduced and well established before it is recognized. Graham and Dunlap[12] have reported that "on a farm where there has been a serious outbreak of swine erysipelas, infection tends to recur in succeeding crops of pigs, either through contact with chronically infected animals that appear healthy or through exposure to contaminated houses, lots, and pastures." The chronic form may also appear on a farm where the acute type has not previously been recognized. New outbreaks do not spread rapidly and widely, as in hog cholera.

The seasonal prevalence of swine erysipelas follows a definite pattern. Gradually increasing from the first to the second quarter, it is by far most prevalent from July to September and declines to the annual low by the end of the year: the approximate percentages from the first to the fourth quarter are 5, 25, 55, and 15. The most susceptible age is from 3 to 12 months, though it is not infrequent in older animals. In Illinois the majority of outbreaks occurred in the first 4 months of age with nearly 16 per cent during the first month (Morrill).[13]

Harrington[14] observed many outbreaks in an acute and highly fatal form in suckling pigs in South Dakota, and Van Es[11] reports that in Nebraska the young, even less than a week old, are sometimes attacked. Evidence of the influence of soil on the viability of swine erysipelas bacilli has been presented by Van Es.[11] It is held that sandy soil rich in lime and humus and ground with an alkaline reaction favor the vitality, while acid soils are unfavorable. Variations in soils are presented as a possible reason for the tendency of the disease to occur in certain regions. Recovered animals are permanently immune.

Bacteriology.—The bacillus of swine erysipelas is a slender, fine, straight or curved rod from 1 to 1.5 microns long. In animals that have died of erysipelas it is readily obtained from the spleen and kidneys, often enclosed in leucocytes. It stains readily with aniline dyes and is gram-positive. Experimental transmission is successful in pigeons, and white mice. Pigeons are highly susceptible. Attempts to produce the disease in swine by feeding or inoculation of virulent material have not been generally successful.

The capacity of this organism to vary widely in virulence and consequently to cause various types of disease has been reported by Van Es as follows[11]: "It has been observed that the bacillus of swine erysipelas may display a capacity to change suddenly from a harmless saprophyte to a decidedly pathogenic parasite. Whether such a phenomenon can be attributed to conditions which exalt the virulence of the microbes *per se*, or to influences affecting susceptibility of the animals concerned, or to some reciprocal action between these two possible factors remains en-

tirely in the dark. Either one of them, however, may serve to explain the sudden increase in the incidence of swine erysipelas in the course of certain years in areas where the malady occurs enzootically."

Distribution in the body, including the blood stream, is general in acute attacks. In subacute or chronic cases the bacilli are found in the affected parts: joints, skin, valvular heart lesions. Healthy swine may carry the bacilli in the tonsils and intestines, and become a constant source of infection of the premises.

Outside the body the bacillus is capable of existence in the soil for at least a year, and under favorable conditions it may multiply there. It resists putrefaction, desiccation and sunlight, and in meat it is not destroyed by pickling, smoking, or drying. To destroy the bacillus by cooking, 2.5 hours boiling are required for a piece of meat 6 inches thick. It has also been observed that certain strains can survive and even multiply in concentrations of phenol that are bacteriostatic or bacteriocidal to many other microorganisms, and that this information has proved to be valuable for isolation of the swine erysipelas organism from specimens where rapidly growing contaminants preclude isolation by the ordinary laboratory procedures—B.A.I. Rep. (1937).[16] In a test of 400 serial lots of hog cholera virus, 4 per cent were found to contain living swine erysipelas oganisms.—U.S.B.A.I. (1944).[16]

Mode of Infection.—Infection enters the body through the digestive tract or the skin. It is probable that it occurs chiefly from ingestion of food or water which has been contaminated by feces from the sick. It may also enter the digestive tract in meat scraps from garbage, and in offal from infected swine that have been slaughtered or that have died. The disease is introduced to noninfected premises by convalescents or carriers, and this mode of infection is increasing in frequency through the promotion of community sales. Often the mode of infection is obscure. It has been suggested that in order to become fully virulent the bacilli must make several passages from swine to swine and that the mere transmission of the specific microbe may be of secondary importance.

Morbid Anatomy.—The pathologic changes of acute swine erysipelas are widely variable and not unlike those of other forms of acute septicemia. Only two pathognomic lesions are recognized: the skin lesions of the urticarial form, and chronic endocarditis. The skin may be normal or extensively purple with edematous thickening in certain areas. The most constant internal lesions are hemorrhagic inflammation of the stomach and small intestine, swelling and congestion of the mesenteric lymph glands and liver, enlarged spleen, hemorrhagic nephritis,

and congestion or hemorrhage of the bladder. The liver is reddish brown. The kidneys are swollen, soft and moist and there may be a few petechial hemorrhages on the surface. The peritoneal and pleural cavities usually contain a small amount of fluid. In general there is absence of hemorrhages in the lymph glands except for a few in the mesenteric group. In the lungs one may find hyperemia and edema.

In the chronic form the most characteristic lesion is verrucose endocarditis with obstruction of the mitral valve. Arthritis is frequent, and chronic inflammatory changes may also be found in the peritoneum and lungs.

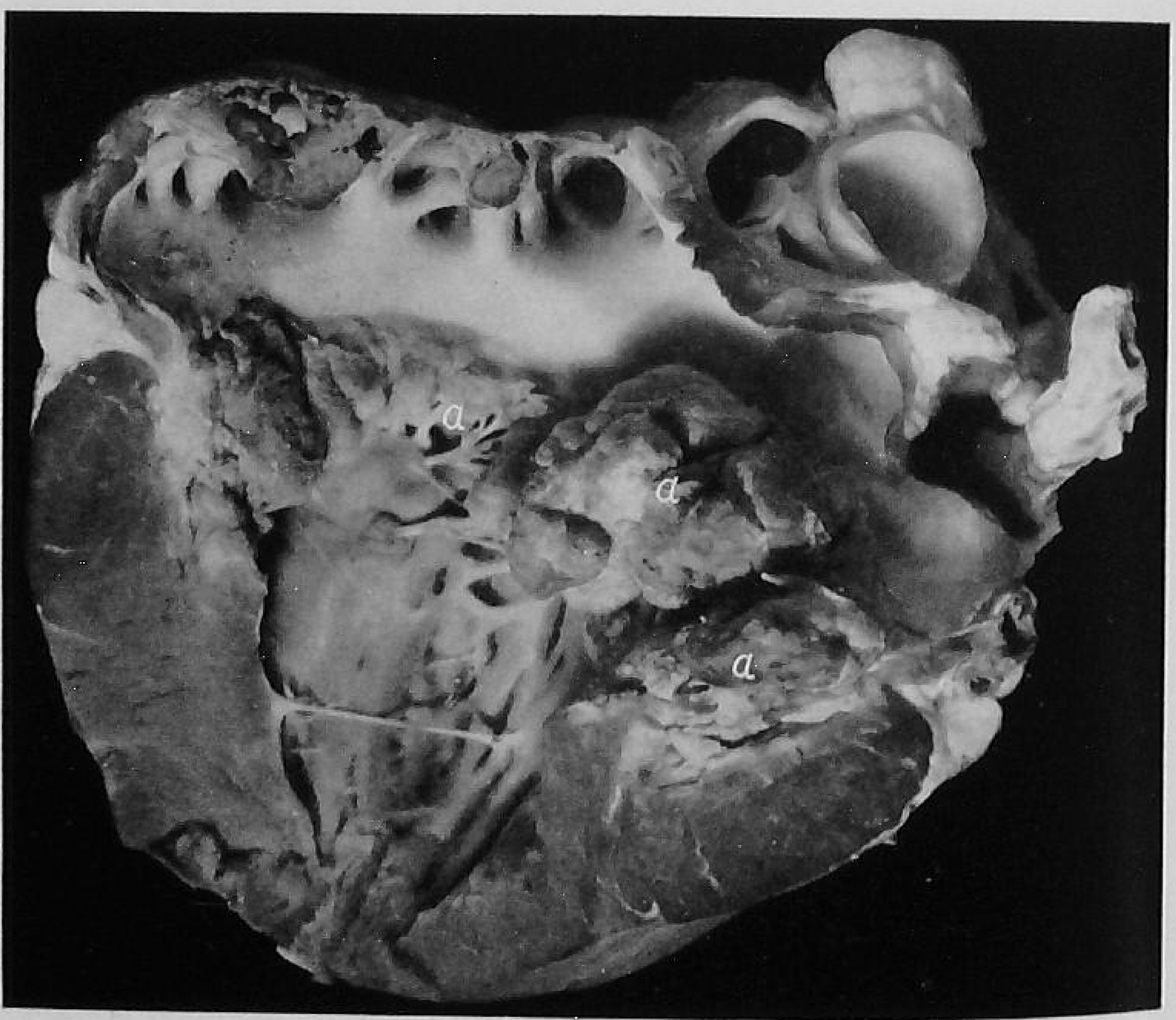

Fig. 73.—Chronic swine erysipelas showing proliferations (a) on the auriculoventricular valves. (Courtesy of D. W. Baker, Cornell Veterinarian, 1933, 23, 66.)

Symptoms.—While the symptoms of swine erysipelas vary widely according to the virulence of the bacillus, the resistance of the animal, and the stage of the disease, two chief clinical forms, acute septicemic and chronic are recognized.

Acute erysipelas is the most important and common form and the

one in which most of the deaths occur. It presents symptoms of acute septicemia with chief localization in the intestines and skin. Several are sick and there is a history of sudden deaths followed by a rapid spread through the herd. In exceptional attacks, however, the spread is not rapid. The period of incubation is from three to four days, the onset is sudden, the temperature varies from 105° to 108° F., and persists until near death or recovery. The sick are depressed, usually they remain hidden in the litter, do not eat, and show weakness of the hind quarters when made to walk. Often there is vomiting. At first there is a tendency towards constipation and this may be followed by diarrhea. When handled or forced to move they squeal with pain. Compared with hog cholera there is less depression, somewhat better appetite and more active response when disturbed. About the second or third day red patches may form on the ventral surface of the body, the medial surface of the hind legs, the throat and ears, and these may fuse to form extensive dark red areas over a large part of the body. While this red color of the skin is regarded as a prominent clinical characteristic, it is not unlike that observed in certain cases of hog cholera. The usual course is from two to four days, though in peracute types death may occur within twenty-four hours. Dyspnea and cyanosis due to heart weakness and pulmonary edema forecast impending death. The mortality rate is estimated by Van Es and McGrath[11] at 50 to 100 per cent with 75 per cent as a probable average. Recorded opinions of the mortality are widely variable. Swine that survive after four days of an acute attack are considered to be possible survivors; this includes the period of highest mortality, but deaths may occur as late as the tenth to the fifteenth day after the onset.

An intermediate or subacute stage has been described by Munce[8] and others; this covers the fourth to the tenth days of survival. The symptoms are extreme weakness and prostration with the hogs usually lying on their sides. After being raised they fall to an unnatural position; if able to walk the gait is stiff, and inappetence is complete. Swelling of the legs, ears, eyelids, and snout are frequent. The skin of the abdomen and other thin-skinned parts may turn red or purple, while the skin of the shoulders, back and sides may become thickened. Multiple arthritis is frequent.

Chronic erysipelas occurs chiefly in the form of an arthritis. It may either follow immediately or be a sequela of the acute type, or it may occur independently. The symptoms are stiffness, swollen joints, and an unthrifty condition. Endocarditis is a sequela of the acute type; the affected animals become dyspneic, easily exhausted and they collapse

when forced to move. Increased heart impulse and endocardial bruits may be heard. Incomplete recovery may leave the animal emaciated and stunted. Stiffness and paralysis are not infrequent. Numerous reports of outbreaks in the United States indicate that arthritis is the most frequent subacute and chronic form, and that often the disease is subacute or chronic from the onset.

Diamond skin disease is the mildest form of swine erysipelas and the type that has chiefly prevailed in the United States. The symptoms resemble those of urticaria. On various parts of the body or limbs there are numerous dark or black quadrangular or rhomboid elevations of the skin. In from one to two weeks healing occurs with sloughing of the epidermis. In severe cases there may be necrosis of the skin with the formation of leather-like crusts. This form of the disease is seldom fatal.

Diagnosis.—Because of the septicemic nature of acute erysipelas, differentiation from other general infections, such as hog cholera and acute Salmonella septicemia, usually cannot be made from the symptoms and lesions alone. In suipestifer infection, however, the age incidence is lower than that usually observed in swine erysipelas. In hog cholera there is a more sudden onset, more complete loss of appetite, a more rapid course, greater uniformity of symptoms, and gluing of the eyelids, which is not observed in erysipelas. In swine erysipelas there is more evidence of pain, sudden unexpected deaths are more common, higher temperatures are observed, and recovery often follows administration of erysipelas antiserum. In hog cholera the lymph glands present darker and wider hemorrhage, they are more generally involved, and multiple hemorrhages throughout the body are more prevalent; the spleen usually shows hemorrhage on the under surface and is normal in size. In erysipelas the spleen is free from hemorrhages, is usually swollen and significance is attached to the diffuse redness of the gastric mucosa. In cholera the lungs are often pneumonic with hemorrhages in the visceral pleura; in acute erysipelas inflammatory lung lesions are rare. The kidneys are involved in both, but in erysipelas the turkey-egg kidney is not seen.

For laboratory examination one may submit spleen, kidney and heart blood from the acute type, and joints, heart, or other affected tissues from the chronic type. Van Es[11] writes that among the diagnostic methods bacteriologic examinations are of the greatest value, because swine erysipelas is the only disorder of swine in which a microorganism of the size and form, and staining characteristics peculiar to the erysipelas bacillus can be demonstrated. Such examinations may be made in the field where smears for staining with Gram's method are made as soon as possible after death from the spleen, cortex of the kidney and

heart blood; in advanced decomposition the red bone marrow may be used. If the bacilli are scarce or decomposition is marked, pigeons may be inoculated with a suspension in physiologic salt solution prepared from a mixture of these tissues. Filter through absorbent cotton and inject 0.5 to 1.0 cc. into the pectoral muscles. Infected pigeons die in from 3 to 5 days and positive smears may be obtained from the spleen, kidney, or heart blood. Schoening, Creech, and Grey[15] have described the technic of rapid-plate agglutination test for swine erysipelas, which has not yet proved to be dependable.

Prophylaxis.—At the beginning of an attack isolate the sick and examine daily for new cases; if possible remove the healthy swine to non-contaminated places and avoid the use of premises where the disease has occurred. Immunization against swine erysipelas is effective. For this purpose one may use serum alone or a simultaneous vaccination. In the United States simultaneous vaccination is now used in highly infected areas under special permits from the Bureau of Animal Industry.

Serum alone has until recently been the only method of immunization of swine in this country. On infected farms one may immunize each new litter of pigs with serum and depend on natural exposure for active immunity. But in highly infected areas where the disease may reappear with each farrowing this method of control may be expensive. Where there is an acute outbreak, serum should be given to all of the swine in the herd. According to Van Es,[11] "public sales establishments, which are apt to have apparently healthy hogs from infected herds assigned to them, may materially protect the interests of their buyers by causing all swine passing through their hands to receive protecting injections of anti-swine-erysipelas serum. No small amount of mischief may be prevented by such a simple and relatively inexpensive measure." The dose of serum is 5 cc. up to 50 lb.; 10 cc., 50-75 lb.; 15 cc., 75-100 lb.; 20 cc. over 100 lb. The duration of passive immunity is from eight to fifteen days.

In the Annual Report of the Chief of the U. S. Bureau of Animal Industry, 1938,[16] the following observations are recorded:

"For the purpose of studying the mode of transmission and methods of control of swine erysipelas, more than 100 hogs have been raised to maturity from a swine-erysipelas-infected herd of 30 sows and boars established last year. These animals were separated from their dams after weaning and placed on clean ground. In none of these animals, now full-grown hogs, has any clinical evidence of swine erysipelas been found.

"Preliminary tests were made with three kinds of live-organism vac-

cines to study the effect of these types of vaccines on the animals. Nine pigs were vaccinated with a product composed of live swine erysipelas organisms of the rough type, which previous study had shown were avirulent for susceptible laboratory animals. Nine pigs were treated simultaneously with (1) a vaccine composed of swine erysipelas organisms of the smooth type virulent for susceptible laboratory animals and (2) swine erysipelas serum. Nine pigs were vaccinated with a commercially prepared live-organism vaccine, such as is used in European countries, and each received simultaneously an injection of swine-erysipelas serum. Each of these groups was maintained in a separate lot with an equal number of unvaccinated pigs. Observations of these animals over a 10-month period following vaccination failed to show clinical evidence of infection with swine erysipelas in any of the vaccinated groups or any of the unvaccinated animals in contact with them continuously. Repeated efforts to develop an experimental exposure to swine erysipelas that would be suitable for testing the relative immunities of these three vaccinated groups have been without success."

Simultaneous vaccination consists in the injection of immune serum and a bouillon culture of the bacillus at the same time. As in vaccination for hog cholera, the injection is made at the base of the ear or into the axillary space: 5 cc. of serum on one side and 0.5 cc. of culture on the other in 50 to 75 lb. pigs. Immunity lasts 4 to 6 months. This method has proved to be highly successful in countries where the disease is widely prevalent. More lasting immunity may be conferred by giving a second injection of the culture about two weeks later. This method of vaccination is now extensively used in a number of states under special permits from the Bureau of Animal Industry (1944)[16] and results obtained in the vaccination of over a million hogs have been very satisfactory. In herds where the disease already exists serum alone should be used in all individuals, followed by serovaccination in from 8 to 10 days.

Double vaccination with Pasteur vaccine consists in an injection of an attenuated culture of the bacillus, followed in about two weeks with a culture of greater intensity. This method confers an active and prolonged immunity, but it is not free from danger; vaccination losses may occur and new centers of infection may be established.

Zwick warns against the danger of vaccination where hog cholera may be latent. As a result, there may be an outbreak of acute cholera. Furthermore, cholera virus may be spread on the injection needle; thus epidemics of hog cholera may follow vaccination against erysipelas. To prevent contamination of serum pour the amount to be used on a

farm from the general supply into a separate container, and sterilize the syringe before using it again on another herd.

Treatment.—*Immune serum* (10-40 cc.) has a curative action when administered early, six to twelve hours after the onset of the symptoms. This may be given subcutaneously, but intravenous or intramuscular administration is preferable. The vaccination of young pigs with 10 cc. of anti-swine-erysipelas serum at 2 to 4 days of age and again at 4 weeks of age practically eliminated enlarged and swollen joints that were becoming prevalent in the pigs in the government herd at Beltsville.—U.S.B.A.I. Rep., 1941.[16]

On the subject of swine erysipelas in Iowa, Aitken[17] writes that when the disease is at a low ebb both prophylaxis and treatment work well, that the disease is variable and unpredictable, and a subject of wide differences of opinion by observers in the Corn Belt. In his discussion on the treatment of swine erysipelas *culture breaks,* for example, he reports that "One client, in May, 1949, had two pigs dead, and two sows and two pigs sick. The pigs were only one to four weeks old, so the entire herd was given erysipelas serum and culture, but 89 days later four pigs again had typical acute erysipelas. Being then old enough the pigs were vaccinated with anti-hog-cholera serum and virus. No more erysipelas vaccine was used yet no new cases have developed on the place since." In another break, 88 days after injection of erysipelas serum and culture in a herd affected with typical acute erysipelas, "we vaccinated for cholera, but this time, as we often do when it seems very virulent, we added erysipelas serum 1 to 6 to the cholera serum. In the handling of serum breaks which occurred in 24 to 36 hours after the use of liberal doses of erysipelas serum and culture in a sick herd, crystalline penicillin, 1000 units per pound of pig, dissolved in a little erysipelas serum, saved 46 of the 49 treated. In another herd the sick were given 100,000 to 250,000 units of penicillin, in 25 cc. of erysipelas serum, to 100 pounds of pig.

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LEPTOSPIROSIS

Definition.—Bovine leptospirosis is an acute general infectious disease caused by the spirochete *Leptospira pomona*. Clinically the symptoms vary widely from mild to severe, of which the most distinctive are depression, fever, and hemoglobinuria with a diminished milk yield from a soft and pliable udder. The milk is yellowish and thickened and may be stained pink or red. Abortion, without other obvious symptoms, is one of the most frequent forms. On postmortem numerous subcutaneous hemorrhages may be found. After recovery, which is the rule, the organism may disappear from the blood and milk while the kidneys may remain infected and spread the infection in the urine.

Etiology.—Leptospirosis in cattle has been reported from at least 30 states with Wisconsin and Illinois leading in the number of cases recorded.[1] While it is often reported that leptospirosis is increasing,[2] it is not yet clear whether the disease itself or our knowledge of it is expanding. In 1944 it was first recognized in this country in Connecticut,[1] in 1948 cultivation of *Leptospira* was reported by Baker and Little,[3] in 1952 a practical diagnostic laboratory technic, complement fixation, was reported by York,[4] in 1953 York and Baker announced the production of a vaccine.[11] Previously, diagnoses were limited to outbreaks where elaborate diagnostic methods for animal inoculation and identification of *Leptospira* were available, or a diagnosis was based on the clinical symptoms. Regardless of these limitations, leptospirosis has been reported from scattered areas in nearly all parts of the country and York[4] has expressed the opinion that there are few

if any regions free from the disease. Infection follows direct and indirect contact with infected animals, drinking water from contaminated pools or sluggish streams, and exposure to urine from cattle or swine with infected kidneys.

After recovery the kidneys remain infected and a possible source of infection in the urine for long periods,[9] in cattle at least 3 months.[4] Since leptospirae are unable to survive refrigerator temperature, it is probable that cattle with chronic infection of the kidney carry the infection from season to season.[9] The spirochete is readily transmissible to guinea pigs, rabbits, mice, embryonated eggs, lactating cows, young calves, and swine. While there is no definite seasonal occurrence, in New Jersey the outbreaks occur more frequently in warm weather. The herd incidence, including animals with inapparent symptoms may be from 50 to 70 per cent.[4] From experimental inoculation of cattle Little and Baker[9] have "recovered the spirochete from the blood, milk, and, occasionally the urine of cows during the febrile period. Moreover, when hemoglobinuria or albuminuria occurred the organism was present in the urine for periods long afterwards. The experimental infections produced in the natural host (subcutaneous or intranasal inoculations) showed all possible variations, from no illness to death. . . . With adult cattle, besides fever, depression, drop in milk yield, and an occasional case of albumenuria, the secretion from the udder was thick, yellowish, and viscid. In lactating cows the inoculation of infective material never produced bloody milk. In young calves, as a rule the infection was acute and associated with albumenuria, or, occasionally, hemoglobinuria and death. On intranasal inoculation, some calves reacted; where others showed no signs of illness. When these animals were later challenged with infective material (subcutaneous inoculations), all were immune."[9]

Recently Burnstein and Baker[10] have reported on infection in swine, where *L. pomona* is often found but where it rarely causes symptoms. "From 17 animals inoculated either subcutaneously or intranasally, leptospirae were found in the blood of 14, isolations having been made as early as the third day and as late as the twelfth day following inoculation. . . . Urines from 34 infected pigs destroyed at varying intervals were tested for leptospirae and all but 3 were found excreting the organism. Leptospirae were seen in the urine of some pigs as early as the twelfth day after inoculation. In two pigs that were kept longest, the organisms persisted in the kidneys and urine of one 159 days after inoculation, and in the other, leptospirae were present in the kidney 140 days after the pig had become infected by exposure. . . . Infection occurred in all calves and pigs exposed to infected pigs. The time be-

tween exposure and demonstration of infection varied from 12 to 45 days. Pigs exposed to infected calves failed to contract infection. . . . All animals inoculated subcutaneously, intranasally, and those exposed by contact became infected. Oral administration of leptospirae was not successful. . . . After an incubation period of approximately 10 days, a transitory elevation of temperature occurred in 22 out of 33 inoculated animals. With two exceptions pigs were free from signs of illness, appeared alert, and ate normally. . . . One to two days after the thermal reaction antibodies were demonstrated in the blood and leptospirae could no longer be isolated. . . . Concurrent with the demonstration of antibodies, leptospirae were found in the urine of all but two pigs that were studied. . . . The leptospirae were usually present in enormous numbers. A serological survey was made of 285 sera collected in large packing houses and 63 (22%) were found to be positive. . . . If the growth and multiplication of leptospirae in the host are criteria, the organism is better adapted to pigs as evidenced by the large numbers excreted in the urine as well as the length of the carrier states. Cattle have not been shown to harbor leptospira for longer than three months. Thus, it appears that swine may be the natural hosts for *Leptospira pomona*."

Morbid Anatomy.—Since the disease is rarely fatal, autopsy reports are limited to the severe form. Thus, Roderick,[6] who described a severe outbreak in Kansas, writes that there were few significant lesions other than bloody urine, the carcass was pale and anemic; subserous hemorrhages and icterus were rare. From an autopsy on an abort that died shortly after birth and in which hemoglobinuria had been observed, subcutaneous hemorrhage and intense icterus were found; in general, the lesions were limited to ecchymoses under the skin, on the heart, and a somewhat yellow abdominal fat.[6] Baker and Little[3] observed that in addition to causing altered milk secretion, the agent damaged the kidneys and produced an interstitial nephritis. In some autopsies white spots 1 mm. in diameter were scattered over the surface of the kidney and extended either as wedges or plugs into the cortical layer. Histological examination disclosed widespread focal involvement of the cortex and corticomedullary junction in the form of irregularly shaped patches of infiltrating cells, predominantly mononuclear.

Symptoms.—Two clinical forms have been described by Little and Baker:[9] "In a *severe* type the onset is sudden with depression, hemoglobinuria, anorexia, dyspnea, a marked drop in the milk yield, and a temperature of 103 to 107° F., and fever persists throughout the illness. In a day or so the visible mucous membranes become pale and icteric. The milk from all quarters is bloody, with a pink, red or

brownish tinge, occasionally containing flecks of blood. The udder is soft and pliable, suggesting little, if any, irritation to the secreting tissue. This limp udder, resembling the gland of a dry cow, is of diagnostic significance. Hemoglobinuria is usually present, the color of the urine being bright red or dark brown. Pregnant animals are quite likely to abort early or during convalescence, and usually the placenta is expelled with the fetus. Before death the symptoms become aggravated. In addition to prostration, the pulse and respiration are increased, the urine is bright red, and the feces are occasionally yellow. If severely affected cattle recover, the convalescence is prolonged due to continued fever, weakness, anemia, and nephritis.

"The mild form is a similar, but less severe, infection, rarely fatal, and lasting for two to four days. There may be depression, anorexia, dyspnea, abortion, and drop in milk yield; or the animal may appear normal except for a lowered production and a change in the character of the milk and urine. A fever of 102 to 105° F. persists for two to three days. The milk may be bloody, but it is more commonly thick, yellowish and viscid. Blood in the milk may be detected at the end of the milking, it may be seen at only a single milking, or it may persist for two or more days. . . . The mammary gland is always soft and limp. Abortions do not occur as frequently as in the severe disease. Hemoglobinuria may occur early or as the cow recovers, and, invariably, the urine is dark brown.

"In some cows, steers, and bulls, the rise in temperature and the hemoglobinuria are the only clinical signs of the infection observed. Occasionally a mild case may die, and animals with hemoglobinuria may not fully recover for weeks."

While this excellent description of the symptoms includes the most uniform characteristics of bovine leptospirosis, the clinical variations from inapparent to severe, have led to widely different reports. Thus hemoglobinuria may be absent, or abortions in advanced pregnancy may occur in a third to a half of the herd, with one or two sudden deaths, and a transient drop in the milk yield.[8] "Bloody urine and yellow milk may be lacking more often than they are present. Often it causes sudden deaths in nursing beef calves about two months old when they commence to drink from the pools." In a beef herd of 360 animals 17 abortions from the sixth to the ninth months occurred over a period of three months; the symptoms appeared in ten days to two weeks after animals were assembled and passed through a chute for dipping or blood testing, and terminated when this practice was replaced by isolation and segregation.[5] In calves, leptospirosis is usually a fatal septicemia with diarrhea. And calves that recover from

an attack at 3 to 4 weeks of age fail to produce antibodies and to react to subsequent serologic tests. Occasionally cows are also affected with the fatal septicemic form. Loses from leptospirosis are measured by interruption with the breeding schedule, lowered milk production, and stunted growth, as well as by the immediate mortality. Mortality may be as high as 10 per cent in adults, and 25 per cent in the young.

Diagnosis.—A diagnosis of leptospirosis in a milking cow can be made when there are fever, hemoglobinuria, and a soft and pliable udder which secretes a yellowish viscid milk that may show a reddish or pink tinge. The presence of leptospirosis in a herd may be determined by a complement fixation test of the blood of the herd,[4] or of the blood of cows that have aborted two to three weeks or more previously. Since the complement fixation antibodies of an animal usually disappear within two to four months after leptospiral infection, this test is of limited value except to indicate more recent infections.

Control.—The problem of control involves a disease in which the clinical cases are usually less than 2 per cent of the infected herd, and the inapparent cases may reach 50 per cent. The attack is limited to one season with little tendency to remain in the herd from year to year. Leptospira has a low resistance and does not survive refrigerator temperature; apparently survival depends on the protection of an animal carrier. It is improbable that it survives for more than a brief period when separated from the protection of a carrier host, such as cattle or swine. Information is not available on the length of time that a bovine remains a carrier and a potential spreader of the infection, but the commonly recommended isolation of newly purchased animals for a period of three months, suggests that it is believed to be transient. The problem of herd control involves protection from exposure to the carrier, as well as to recently contaminated food or water with which the carrier has been in contact. There are numerous reports of cessation of the disease following a change of pasture, or herd management or suspension of outside contacts. Little has reported repeatedly that experimental animals, including the sick and the inapparent infections were immune when challenged later by subcutaneous inoculations. And while the vaccine developed by York[4] has not been tested extensively in the field, apparently it confers protection against contact exposure. Until more information is available vaccination of negative animals in infected herds is advised. Purchased additions from unknown sources should be segregated for 90 days.

Treatment.—Severe acute types should be treated as other acute general infections. Among the antibiotics that have been favorably reported is terramycin, 2 mg. per lb. body weight per vein daily until

improvement develops; or procaine penicillin 1.5 to 3 million units intramuscularly every 24 to 48 hours; or streptomycin, 5 mg. per lb. body weight intramuscularly every 12 hours. As a measure of control it is advised that in infected herds calves be vaccinated soon after birth and again in six months, and that mature animals be vaccinated yearly.

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VIRUS DISEASES

HOG CHOLERA

Definition.—An acute general infection caused by a filterable virus, characterized anatomically by hemorrhages in the skin, kidneys, bladder, lymph glands, and spleen. In the beginning of an outbreak the postmortem changes may be slight or absent. Clinically the disease is marked by higher fever, and prostration. Early in an outbreak in a herd several deaths may occur, almost without warning.

History.—In the United States the first recorded outbreak of hog cholera was in Ohio in 1833. During the next 60 years a destructive contagion of unknown cause among swine was common in the Middle West. After about 1850 this became the subject of various papers and reports from Detmers, Law, and others.[1] They recognized it as a specific contagious disease and described the symptoms and lesions. In 1885 Salmon and Smith[2] reported extensively on the symptoms and lesions. According to their interpretation of this malady, there were two independent diseases: swine plague, caused by *B. suisepticus*, and hog cholera, caused by *B. suipestifer*. In 1903 the specific cause, a *filterable* virus, was discovered by DeSchweinitz and Dorset[3] and this led to the production of a protective serum in 1908.[4]

Eitology.—*(a) General Prevalence.*—Dorset and Houck[5] report three periods of exceptional prevalence: the first reached its height in 1887, the second in 1897, and the third in 1914. They estimate the average annual loss for a period of 40 years at not less than $30,000,000. In the United States, in 1925, nineteen states containing approximately 77 per cent of the total number of swine, suffered losses of from 2.4 to 6.5 per cent; all except California were in the Middle West or South. The remaining 23 per cent of swine were distributed over twenty-nine states, and the mortality was from 0.6 to 2.2 per cent of the total number of animals. Thus there are large sections of some states that have no hog cholera, though none is reported as entirely free. While it often occurs at any period of the year, its chief *seasonal occurrence* is in the late summer and fall, especially in October and November, but in the South severe outbreaks may occur at any season of the year, and it is here that the highest death rate occurs. *Pigs from immune sows* are born with an increased resistance, though infrequently they begin to contract cholera when less than a month old (Pickens).[6] In Canada small outbreaks are occasionally caused by feeding uncooked garbage or by the importation of swine from the United States, but widespread

infection is prevented by prompt segregation, slaughter, and the use of serum. It is common in Europe.

(b) Infected Swine.—The diseased herd is one of the chief sources of infection. It is a fundamental law of hygiene that when an acute contagious disease appears, the infected and exposed individuals should be segregated; but when hog cholera appears in a herd it is a common practice to rush all apparently well animals to market. This infects traffic routes, spreads infected pork scraps wherever the finished product is marketed, and infects other herds when the survivors are reshipped for additional feeding. On this subject the U.S.L.S.S. Association[7] recommends that "the shipping of infected hogs to public markets be discouraged. Realizing that the sick hog is the original source of infection, and that its meat may carry active virus, federal and state regulations should impose severe penalties in the way of condemnations when consignments of hogs visibly affected with cholera arrive at public markets." In 1931 the Committee on Transmissible Diseases of Swine reported: "This year, as in previous epizootics of hog cholera, large numbers of cholera-infected hogs were marketed. Important factors in the dissemination of hog cholera are the marketing of these hogs for slaughter, and the movement of stock hogs from communities in which these early outbreaks of the disease occur to live stock centers and community sale barns, and to farms and communities that are free, or comparatively free, of cholera infection. Failure to enforce regulations or to pass laws to meet the conditions responsible for hog cholera epizootics, accounts for a large part of the financial loss resulting from this and other diseases of swine. The use of trucks in the transportation of swine, as well as all classes of live stock, should receive additional attention in the control of their operation as a common carrier of live stock."

The introduction of infected hogs was responsible for 44 per cent of all new or primary outbreaks in eleven middle western states, according to returns on a questionnaire reported by Atherton.[8] In Maryland this accounted for only 6 per cent. From the primary outbreak infection spreads in many ways, both by direct and indirect contact, and the more dense the swine population, the more rapid the spread. The virus may be carried on the feet of farmers, hired help, visitors, and stock buyers; or it may be brought from infected farms in litter, on machinery, wagons, and trucks. Dogs, birds, and other scavengers scatter parts of unburied carcasses. Direct contact occurs when swine break through a line fence, or are taken away for breeding, or for exhibition. The virus may be carried in streams or drainage. Harbored infection is also recognized, though we have little knowledge of the length of time

that virus may retain its virulence on infected premises under favorable conditions. On one farm in our experience it apparently remained in a stable for at least two years. Under natural conditions the disease spreads rapidly, yet efforts to transmit the disease by experimental contact have usually been negative—Dorset.[9]

(c) Feeding Raw Pork Scraps.—In 1912 McGilvray[10] reported an outbreak of hog cholera in Canada caused by the feeding of infected market pork. In 1917 Birch[11] published the results of an experiment which showed that "in places where meat inspection is maintained, it is impossible, even with the severest interpretation of temperatures, symptoms, and lesions now practicable, to remove from market all carcasses of hogs that contain hog cholera virus." In an examination of 21 sugar-cured hams from infected no-lesion hogs, virus was found in 12. This was found to persist for as long as eighty days. It is probable that in the eastern states from 85 to 90 per cent of new outbreaks can be traced to virus in pork scraps fed in garbage.

(d) Simultaneous Vaccination of Devitalized Swine.—During the first few years after the discovery of immunization with serum and virus, the simultaneous method was considered superior to the use of serum alone under all circumstances—(Koen).[12] But repeated heavy losses from so-called "breaks" have led to the belief that the use of serum and virus in devitalized hogs is a frequent cause of new outbreaks. This experience has found expression in reports of the Proceedings of the U.S.L.S.S.A., as follows: "What is to be done in communities where there is hog flu, extensive parasitism, and necrotic enteritis? Do not give simultaneous treatment. Give serum alone until in condition to receive other treatment"—Houck, 1927. "Questionnaires received from veterinarians of eleven western states show that abuse of the double treatment was the second largest factor in starting outbreaks of cholera in that territory. In fact, the data derived from these questionnaires show that approximately 40 per cent of the new outbreaks were started in this manner"—Atherton, 1930. "The administration of serum and virus to hogs exposed and suffering from disease other than cholera accounts for many so-called 'serum breaks' and a large part of the death rate subsequent to vaccination"—Committee Rep., 1931. Others are of the opinion that many "breaks" have been caused by the use of impotent or insufficient serum.—Benner.[13]

There has been considerable discussion of the cause of deaths from "breaks" following simultaneous vaccination. One group defends the method of vaccination and maintains that losses result from some disease other than cholera. A second group condemns this method of vaccination in "devitalized" swine, and apparently believes that deaths

following such practice are due to cholera. A third group holds that "breaks" following simultaneous vaccination are cholera due to impotent serum; it may have lost its strength through age; it may have been defective in origin, or the dose may have been too small. Breaks may also be due to impotent virus. At present, most workers on diseases of swine accept the second view. When a hog sickens and dies from the injection of cholera virus, it is illogical to attribute death to "ascariasis," "flu," "mixed infection," etc. It is equally illogical to inject cholera virus into swine that are obviously sick of cholera or any other disease.

(e) The Virus.—Inside the body a filter-passing virus is found in all the body fluids and tissues early in the incubation period, and this virus is usually present throughout the course of an acute attack. In the chronic form the blood may be found free of virus, and there is no knowledge that it is carried by recovered swine. It is harbored and eliminated before symptoms may be recognized. All discharges from the body openings of the sick carry the virus and it is especially abundant and virulent in the urine. After swine have received virus for immunization, they should not be added to susceptible herds until at least three weeks have passed without any signs of disease.

Outside the body the vitality of cholera virus is not well known. According to Birch,[11] "it is very tenacious and resists most natural destructive influences for a long time." Kernkamp[14] found the virulence of hog cholera virus is not destroyed or lost up to approximately three years of age, if it has been carbolized and kept under low temperature conditions. Its survival in pork scraps was found by Birch[11] to be as long as eighty days. It succumbs quickly to putrefaction. Hog wallows are thought to remain infected for months, and it is probable that in exceptional cases the virus may retain its vitality for years.

Morbid Anatomy.—Hemorrhages are the distinctive lesions of hog cholera; these are widely distributed in the serous and mucous membranes, skin, lymph glands, and internal organs. As in other acute septicemias, some of the first to die in an outbreak may show no changes on autopsy. But in the usual *acute* form the postmortem changes clearly reveal the nature of the disease. The *skin* may show slight or extensive hyperemia on the ears, abdomen, and other parts of the body; sometimes there are small areas of necrosis at the margins of the ears and vulva. On section the *lymph glands* show peripheral hemorrhage, petechiae and ecchymoses, and they may present a dark, almost black, surface. Various degrees of hemorrhage are commonly found in the visceral peritoneum, on the under surface of the spleen, in the mucosa of the digestive tract, especially in the region of the ileocecal valve, in the large intestines, and in the lungs. Of special

significance in diagnosis are petechial hemorrhages beneath the capsule of the kidney; these are rarely absent in cholera and their presence is almost pathognomonic of this disease. Of equal significance, though less frequent, are hemorrhages on the mucosa of the bladder, and purple discoloration of the skin. A fibrinous exudate may be found on the serous surface of the stomach, and erosion is sometimes met with in the large intestines. In the occasional *chronic* form these erosions have become ulcers—"button ulcers"; they are raised above the surface, and are dark or yellowish in color. It is probable that the presence of "button ulcers" is due to chronic infectious enteritis more often than to chronic hog cholera.

The changes found on postmortem examination in chronic cholera are emaciation, shrunken spleen, ulcers in the mucosa of the bladder, and button ulcers in the large intestines. Hemorrhages are absent. Various degrees of pneumonia and pleuritis are frequent.

Symptoms.—The period of incubation is 5 to 10 days. In the *peracute* form the animals die without showing recognizable symptoms. The *acute* form begins with chills and a temperature of 105° to 108° F.; the temperature continues and fluctuates until near the end, when it is subnormal. In a recently infected herd a few have died, some are obviously sick, and others are apparently well. At first only a few are attacked, but the infection gradually spreads and in a few days all are either exposed or sick. The general symptoms are marked depression or prostration, refusal to eat, stiffness, disinclination to move, head down and tail hanging straight; often the pigs hide in the litter, or in cold weather pile in heaps. Occasionally, in advanced cases, purplish hemorrhagic patches appear on the ears and abdomen; these are seen only in hog cholera and swine erysipelas. The margins of the ears, the tail, and the lips of the vulva may show small necrotic areas. Thumps and cough are occasional. Forced exercise causes acute exhaustion and prostration. Conjunctivitis is often present. Constipation followed by diarrhea is common. Motor irritation may be noted in the beginning in the form of circling, muscular twitchings, and even convulsions.

In the acute form the course is from five to seven days and the mortality is from 85 to 100 per cent. Survivors may recover completely or suffer from a subacute or *chronic* form. In the latter, infection localizes in the lungs or intestines, causing cough and dyspnea or diarrhea; emaciation develops and the victim finally succumbs or becomes worthless. In the early stages of cholera there is a marked decrease in the leucocytes (leucopenia).

The *diagnosis* of hog cholera usually is not difficult when the history is available, and the symptoms and lesions of typical acute cholera

are present. A diagnosis of hemorrhagic septicemia should not be made until the disease is proved by filtration experiments not to be cholera; most epidemics of septicemia are cholera. Acute swine erysipelas closely resembles cholera, and the disease is now present and apparently spreading in the United States. As a final test one may inject a pig susceptible to cholera with filtered blood from a sick animal. When sudden deaths occur, and autopsies are negative, postpone the diagnosis until positive evidence is available. The symptoms in the early stages of necrotic enteritis may closely resemble those of hog cholera.

The mere fact that several individuals are sick is highly suggestive. Learn if there has been an opportunity for exposure through feeding of garbage, new arrivals, or hog cholera in the vicinity. Note the season with reference to the high prevalence of the disease in the fall and early winter, and the year with reference to general prevalence of the disease. The significant general symptoms are the acute fatal course, chills, purple discoloration, depression, high fever, and conjunctivitis. Then there are distinctive localizations, as hemorrhage and necrosis of the skin, or vomiting and diarrhea. The most useful diagnostic evidence comes from an autopsy. This consists in the finding of peripheral hemorrhages in the lymph glands, hemorrhage in the mucosa and serosa, petechiae on the kidneys, on the spleen, and on the mucosa of the bladder. Kidney hemorrhages are nearly always conclusive. The first to sicken may die quickly and fail to show lesions on autopsy, and a failure in diagnosis is easily made by drawing definite conclusions from negative evidence. The differential diagnosis of swine erysipelas and hog cholera is described under the subject of swine erysipelas.

Prophylaxis.—*(a) Immunization.*—The record of vaccination as a *cause* of hog cholera indicates the need of complete knowledge of the herd for which *prevention* is desired. In discussing the element of danger caused by the injection of the virus of cholera, Dorset and Houck[5] state, "If the serum should not be of proper *potency* or a sufficient *dose* is not administered or if the *work* is not done properly, hog cholera may be produced." A survey of recently published reports (see references under etiology) indicates that numerous other causes of failure may be added to potency, dosage, and work.

Two methods of immunization with antiserum and virus are available: serum alone, which confers passive immunity lasting two to six weeks; and serum-virus, which confers permanent immunity in properly selected swine. In the use of the simultaneous (serum-virus) method, one must consider that the hog is protected against the virus in part by the serum, and in part by its own natural resistance. In every case the virus causes some reaction, though this may not be apparent. If

natural resistance is lowered, as from exposure, transportation, or parasitism, the virus may overcome the protective action of the serum and cause cholera within a few days; this is termed a "break." Formerly breaks were attributed to inert serum or virus, but now they are chiefly explained by the vaccination of "devitalized" hogs. If the virus becomes inert, cholera may appear within a few days to a few weeks following simultaneous vaccination; such breaks have occurred repeatedly in New York State.

Following the vaccination of apparently healthy swine, the appearance of a septicemic disease resembling cholera indicates either the failure of immunization, a "break," or the presence of some other septicemic disease, perhaps swine erysipelas, or pig typhus. The differential diagnosis of swine erysipelas and hog cholera is described under the subject of swine erysipelas. In post-vaccination breaks, *S. suipestifer* is being increasingly incriminated by both research workers and practitioners. Thus Van Es and Olney write that "The causative microbe of pig typhus is responsible for a relatively high morbidity rate among juvenile swine and apparently it has an ubiquitous distribution in farm hog herds. It would therefore be difficult to exclude it as a potential contributory factor in the etiology of so-called breaks. . . . It is quite possible that much mischief may be attributed to the exaltation of the virulence of *S. suipestifer* by the simultaneous presence of hog cholera virus, rather than to the cancellation of hog cholera immunity on the part of the pig typhus bacillus." *S. suipestifer* bacterin has been used previous to vaccination with serum and virus in areas where outbreaks of necrotic enteritis frequently follow the injection of hog cholera virus. While the value of such vaccination may be questioned, it is stated in the 1943 U.S.B.A.I. Report that "the bacterin treatment shows promise of immunizing value." Anti-swine-erysipelas serum may be combined with hog cholera vaccination.

The following specifications for the safe use of serum alone, or for serum and virus, have been formulated by Birch[15] and others:

The simultaneous method is indicated: (1) In sound herds where infection is bound to occur, but may be delayed; (2) in sound herds on infected farms; (3) on farms where there is a history of cholera; (4) in large herds where hogs are frequently bought and sold; (5) on infected farms with a reasonably large enclosure, those showing a temperature below 104° F., and no symptoms; (6) in garbage-fed hogs; (7) in swine weighing not less than 40 to 50 pounds; (8) sows in early pregnancy; (9) show hogs a month before they start, either isolate the hog to be immunized or inject the entire herd. Before injecting virus

take the temperature of each animal; an apparently normal herd may contain many individuals with a cholera temperature of 106° F. or more. After vaccination provide clean dry quarters and feed on a restricted diet for two weeks. Do not allow contact with nonvaccinated or nonimmune swine for at least three weeks.

Serum alone is indicated: (1) In young pigs four to five weeks old; (2) where hog cholera has started; (3) in sows that are weaning pigs; (4) when an infectious disease is present; (5) when the herd is unthrifty; (6) in hogs not accustomed to present surroundings and food; (7) when the entire herd cannot be immunized; (8) when the herd cannot be isolated; (9) during weaning and castration; (10) when exposed to filth and no shelter; (11) when about to ship from stock yards; and (12) sows in advanced pregnancy.

Birch[16] gives the following directions for immunizing *young pigs:* "In garbage-fed herds, and in other herds where there is constant danger of infection, serum alone should be administered when the pigs are from four to six weeks of age, and sooner if hog cholera should develop. This should be followed by simultaneous treatment when the pigs are from nine to twelve weeks of age. In the present state of our knowledge twelve weeks is greatly to be preferred, but if the herd begins to 'break' before this time, serum should be administered at once. In case it becomes necessary in any particular herd to administer serum when the pigs are two or three weeks of age, a second dose of serum should be given some four weeks later, and this followed in the usual manner with simultaneous treatment when the pigs reach an age approximating twelve weeks."

Because of the brief period of immunity produced by serum alone, two to six weeks, and the cost of repeated injections, its use has been limited. In the important swine-raising states the simultaneous method has been used almost exclusively. At best, vaccination is merely a system of insurance against the disease. It does not limit the area of infection. In the period 1928 to 1940 there was an annual loss from hog cholera of 2.5 to 3 per cent of the total swine population of the United States.

Technic.—In handling serum or virus, a 5 per cent solution of phenol, or its equivalent, should be poured over the mouths of the bottles before they are emptied. Provide a covered sterile container for serum and one for virus, or remove from a stoppered bottle through a canula. Use a 50-cc. Record or similar syringe with a 16-gauge 1.5 inch needle for the serum and a 5 to 10 cc. Record or similar syringe for the virus. Inject one-half the serum in each axillary space, except in large animals, when

it is injected behind the ear. Inject the virus in the chest muscle. The field of operation should be thoroughly cleansed and disinfected. Care is needed to estimate accurately the weight of the hog, and to give no less than the dose printed on the container. In unthrifty individuals the dose of serum may be doubled.

Crystal-violet vaccine in the control of hog cholera has been distributed by the U.S.B.A.I. (1942-45), and its use is increasing. In Great Britain it has been adopted to the exclusion of all other methods. In a large-scale trial at the Weybridge Veterinary Laboratory, injection of this vaccine into susceptible pigs of any age over 3 weeks did not cause illness or abnormality and produced an immunity of high order for about 12 months. The dosage is 5 cc., and immunity is said to be adequate by the twenty-first day. Since it is a sterile product its use removes the suspicion that a "break" may have been caused by the use of virus. Compared with serum and virus it is less expensive and it does not cause either reactions or disease, but the development of immunity is more gradual.

Boynton's tissue vaccine (B.T.V.) is a suspension of finely ground virus tissue collected from pigs in the acute stages of cholera infection. Like crystal violet vaccine, it cannot produce cholera when injected into healthy pigs, and there is no reaction following its use in pigs suffering from intercurrent infections. Immunization requires a period of at least three weeks. It is claimed by Boynton[17,18] to be an adequate and safe immunizing agent against hog cholera and one that can be used without fear of aggravating intercurrent infections or spreading hog cholera infection.

In the summers of 1949 and 1950, serious postvaccinal losses have drawn repeated editorial comment in the Journal of the A.V.M.A.[19] A high percentage of the affected pigs sickened during the "reaction" period, usually from the sixth to the tenth day, and most of them died. The most widely accepted explanation of this disaster was that a variant virus or viruses had contaminated certain series of immunizing virus, and this opinion has been confirmed by the U. S. Bureau of Animal Industry.[20] By increasing the dose of serum 50 per cent, the ill effects of the virus could be reduced or eliminated. Pigs vaccinated after weaning were more susceptible to the effects of the variant virus than were suckling pigs. The crystal violet vaccine and Boynton's tissue vaccine are products of an effort to avoid the danger of virus, but their action is limited by the slow development and uncertain duration of immunity. A more recent type of vaccine, the *modified or lapinized virus vaccine,* became available in September 1949, but "Of those who have used them, a high percentage has been disappointed by post-

vaccinal trouble. It is hoped that further improvement of the modified vaccines, and further experience in their field use may develop them to a point where they will be both safe and effective."[19]

Sanitation.—After the infection has passed, pens should be disinfected and cleaned, while fields, lots, and pastures should be drained. A group of hogs may be protected against infection, even when it is near, by confining them in pens from which all other animals are excluded. Provide a tub of disinfectant in which the caretaker may dip his boots before entering. When cholera appears in a swine-raising locality, each owner should take precautions to protect his farm against carriers. Dead animals should be promptly buried in lime or burned. Change pasture frequently. As in most diseases caused by specific infection effective control depends on closing the channels through which infection spreads, such as the feeding of uncooked garbage, transportation of infected swine, and indiscriminate use of virus.

Treatment.—Serum has been extensively used in the treatment of cholera, but usually without success. Serum given in double dosage in the incubative stage, or in the beginning of the attack, may sometimes be beneficial. When a break occurs among vaccinated pigs, prompt use of serum may prevent heavy loss. But if this is not used until half the animals are sick it is of no value.

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INFECTIOUS EQUINE ANEMIA

(Swamp Fever; Recurrent Fever)

Definition.—Infectious anemia is an acute or chronic septicemic disease of solipeds (horse, mule, ass) caused by an ultramicroscopic specific virus and characterized by recurrent fever. The specific cause was reported by Carré and Vallée[1] in 1904. It is unlike pernicious anemia in man.

Etiology.—The disease was first described by Lignée in France, in 1843. In North America it was probably observed in Manitoba in 1880. Since 1900 it has been widely recognized in certain well-defined areas over a large part of the United States west of the Mississippi River and in certain parts of Western Canada. In 1915 it was identified by Udall and Fitch[2] in Northern New York, where it had prevailed at intervals for at least twenty years. Since 1920 its prevalence in the United States has apparently diminished. In Europe, Russia, Japan, and Africa the disease is widespread. In 1929 the disease was reported from seventeen states, and in all except Mississippi, Arkansas, and Idaho it was regarded as of little economic importance. In the Delta region of the Mississippi it constituted a major economic problem (Stein).[3] It is favored by soil that is moist and poorly drained and by wet seasons when biting insects are numerous. It occurs chiefly in the summer and reaches its height in the acute form during midsummer.

The prevalence of the disease in moist locations, as on the Coastal

Plains of Texas, and in the lowlands of the Platte and Mississippi rivers, has given to it the name of swamp fever. It is not infrequent, however, in higher altitudes and on dry soils where water contamination cannot be suspected. Fulton[4] observed that in Saskatchewan the disease never occurs in the heavy clay lands, and where it appears with regularity the soil varies from a heavy to a sandy loam. He associated the disease with the drinking of water from sloughs where vegetable growth is plentiful. The majority of acute and subacute cases are seen in the months of July to September. Chronic cases are seen in the winter and spring, and they are probably the result of infection which took place in the previous summer and fall. It is a pasture disease that tends to localize on certain farms, and, according to Scott,[10] is never contracted in the stable.

The ultravirus is found in the blood of diseased and recovered horses. It is constantly present in the urine and eye secretions, and may be found in the feces, saliva, and milk. Schalk and Roderick[5] have reported "that an artificially infected swamp fever case may survive the disease for fourteen years without anemia and then without any exciting factor rapidly break down, showing all of the phenomena of a typical case, including profound anemia." It has been observed repeatedly that in infected areas carriers may be so frequent that horses on swamp-fever free areas or farms are the only ones suitable for experimental inoculation with suspected material. The virus has been demonstrated by Lühr[6] in *Gastrophilus* larvae, filaria, biting flies and mosquitoes. Outside the body it may remain active for weeks and months in contaminated bedding and straw. In experiments reported by the Bureau of Animal Industry,[7] the disease was transmitted by injection of extracts prepared from *Strongylus* spp.[7] adult worms, and by milk secretion from an infected mare; and blood collected from a newborn foal before it had nursed the infected dam was found to contain virus. Attempts to transmit the disease by urine were negative, but evidence was obtained that semen[8] of an infected stallion contained the virus. Transmission to the offspring in utero has been reported by Stein and Mott.[9]

There are two theories concerning the mode of infection. Experimental transmission was first done by Carré and Vallée[1] in 1904 by feeding blood. This led to the belief that infection results from ingestion of contaminated food and water. This view is supported by experiments conducted by the Bureau of Animal Industry (1938)[7] in which the disease was transmitted by intimate stable contact[7] in which the animals intermingled unrestrained in a large stable, drank from a common trough, and fed from the floor. Insects were excluded by screens. The disease did not spread from infected to normal animals when they were

kept in adjacent stalls or adjoining box stalls, and water and feed were supplied from individual containers. Experiments by Scott[10] have demonstrated that infection is transmitted by bites of stable flies, *Stomoxys calcitrans*, and the gadfly, *Tabanus sepentrionalis*, a mode of conveyance in agreement with the epidemiology. It may also be transmitted by mosquitoes.[11] Fulton[4] has reported experimental production of swamp fever by intravenous injection of slough water. This observation supports the theory of Lühr,[6] that natural infection may take place through the ingestion of food and water that has been contaminated by infected insects. Infection may also be carried on hypodermic needles and other instruments; it does not usually take place by direct contact and equines are the only susceptible animals.

The virus resists putrefaction, drying, and freezing, and is killed by exposure to heat at 140° F. for one hour. In dried blood the virulence may persist for seven months. The infection spreads slowly. It has been observed that when susceptible northern mules are shipped to infected areas in Mississippi the disease does not appear among them until the second year. Evidence indicates that the horses of a given locality grow tolerant after several years and that the prevalence tends to diminish. Where conditions favor transmission the spread of the disease follows the introduction of infected horses. Thus there was a marked increase in prevalance in Central Europe during World War I; this was attributed to the introduction of carriers from Russia on the east and France on the west. While many animals appear to be resistant, there is no certain immunity.

Experimental transmission is most readily accomplished by subcutaneous or intravenous injection of infected blood. The period of incubation is from one to two weeks, irrespective of the amount used. The Japanese Commission[12] obtained infection in thirty days after feeding urine (100-200 cc. daily). Complete resistance exists in cattle, sheep, goats, swine, dogs, cats, mice, rabbits and guinea pigs.

Morbid Anatomy.—Acute infectious anemia shows the postmortem changes of septicemia: marked enlargement of the spleen, enlarged liver, hyperemia of the lymph glands, and hemorrhages throughout the body. In the heart are extensive subserous and subpericardial hemorrhages, from petechiae to bloody suffusions, and ecchymoses may be found within the heart muscle. The *chordae tendinae* and valves are sometimes edematous. In the heart muscle, beneath the endo- and epicardium, and in the intima of the aorta are grayish or yellowish white foci; these have been regarded as possibly specific lesions of swamp fever. The heart muscle is degenerated. Petechiae are often present on the surface of the liver, and they are usually present on the surface of

the lungs. The kidneys may contain small abscesses in the cortex, and there may be parenchymatous degeneration; often the external surface appears normal. Subserous and submucous hemorrhages are abundant in the intestines. The abdominal cavity contains varying amounts of reddish fluid, while the peritoneum may be either congested or hemorrhagic. While the bone marrow is intensely red from regenerative activity, this change is not specific of infectious anemia.

In *chronic* forms the postmortem changes may be limited to anemia and emaciation.

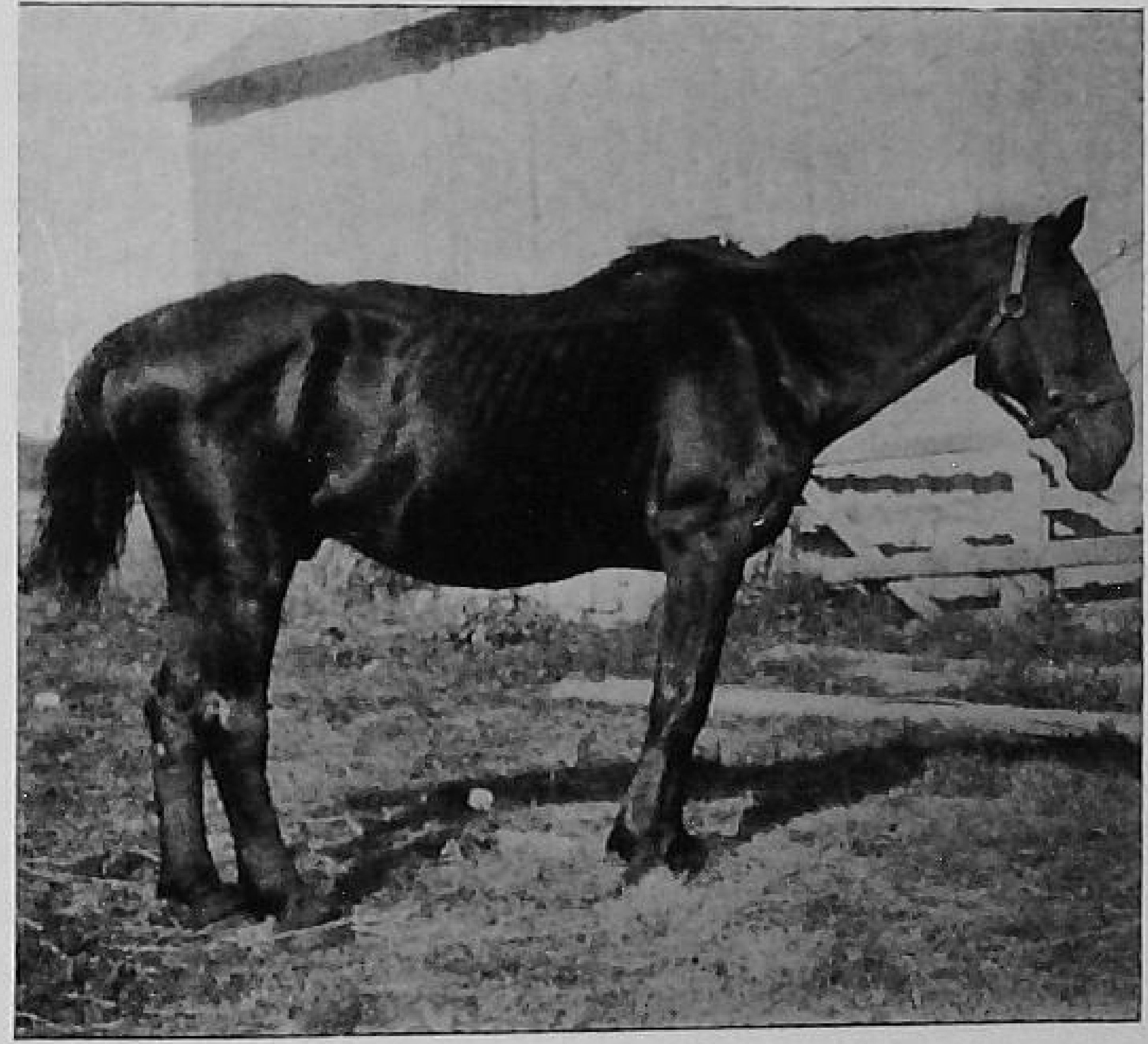

Fig. 74.—Infectious equine anemia.

Symptoms.—The period of incubation in experimentally produced swamp fever is from seven to twenty-eight days—Stein.[3] Three clinical forms of infectious anemia have been described: acute, subacute, and chronic. Cases representative of each type are frequent, yet there is no sharp line of demarcation. Because of the cyclic occurrence of infectious anemia, and apparent tolerance where the horse population is relatively fixed, there are wide variations in the symptoms and course. The terms acute, subacute, and chronic, apply to the intensity of the syndrome, as well as to the course. An acute attack may terminate fatally in three days, or an animal may have a series of acute attacks and apparent recoveries over a period of weeks and months. In the final and fatal attack, the symptoms are usually acute. Where conditions

favor transmission, and where there are many susceptible horses, acute manifestations and remissions are relatively frequent. For example, in the outbreak observed by Udall and Fitch,[2] horses frequently made apparent recoveries, but the final mortality was approximately 100 per cent. Where the disease is regularly endemic chief losses are from debility and not from death.

An *acute* or *peracute* attack may continue for from three days to three weeks. It is characterized by sudden and marked prostration, high continuous fever (104° to 107° F.), reddish icterus of the conjunctival mucosa and slight or distinct edema of the subcutis of the limbs or ventral parts, particularly the prepuce. Petechial hemorrhage may be observed on the membrana nictatans. There is a definite stiffness, sometimes suggestive of tetanus, that apparently is caused by weakness. There may be a fair appetite, but the horse eats slowly. A slight serous discharge from the eyes or nose is not infrequent, and the nasal discharge may be blood-tinged. Drooping ears and half-closed eyes mark the more severe attacks. Often the pulse rate is relatively much lower than the temperature. Towards the end the pulse is high and the breathing becomes labored. Polyuria and albuminuria are usually present, and there may be a moderate diarrhea. Recurrent attacks are not infrequent; when the intervals between attacks are only a few days the disease soon ends fatally.

The *subacute* form presents similar but less intense symptoms. The course is over a period of four to eight weeks, and is marked by apparent recovery followed by remissions. Exertion causes dyspnea, pounding heart beat, sweating, and exhaustion. Usually death occurs during an acute phase, but the attack may become chronic. The temperature is somewhat lower than in the more acute form and some observers report finding a pale, instead of an icteric, conjunctival mucosa.

In the *chronic* form, anemia and intermittent fever, loss of condition, dullness, and sometimes jaundice may continue for months. The symptoms may be slight or they may be entirely absent (latent) for months at a time. Such cases tend to break down when worked. Where the disease is permanently endemic and relatively frequent, there may be many carriers or latent types.

Blood examination discloses definite changes, even in only slightly affected individuals. When the oral mucosa is cut for the purpose of taking a blood sample, the blood may be serous and fail to clot. The hemoglobin may be as low as 30 to 60 per cent, while the erythrocytes may drop from 7 or 8 millions to 2 or 3 millions. On the other hand the blood may remain normal. Van Es et al.[13] state that "many cases of swamp fever terminate fatally without a marked reduction in the red

blood cells, a fact denying the popular conception of 'swamp fever' being primarily an anemia." Lühr[6] also reports that in some patients the erythrocytes are normal in number until death.

Diagnosis.—Because of the high mortality and the existence of latent infection and carriers, the diagnosis of infectious anemia is highly important. A blood inoculation of a susceptible horse is the only method of making a positive diagnosis, and in an artificially infected horse the disease may be latent. In New York State it has been confused with poisoning from eating hay contaminated with *Equisetum* (horsetail). Steck[14] comments on the peculiar and unexplained occurrence of swamp fever in certain restricted localities, and the frequency of only one sick horse on a farm, as commonly observed in Switzerland. He attaches important clinical significance to the presence of 50 or more sublingual hemorrhages which he has observed in the sick and in their apparently normal stable mates, and advances the theory that by the time a horse becomes obviously sick all of its stable mates are infected but not sick, and that infection usually is light with a tendency to spontaneous recovery. Similar sublingual hemorrhages were not found in other infectious diseases. Steck also was able to induce sublingual hemorrhages, without other symptoms, by intravenous injection of filtered serum from an apparently normal carrier of such hemorrhages, and by subcutaneous injection of filtered serum from convalescents. These hemorrhages are described as ¼ to 1 mm. in diameter, sometimes larger, superficial, located on the under surface of the tongue, and more abundant towards the tip. Stein et al. have also reported that infectious anemia in an inapparent (subclinical) form can be induced in a susceptible horse by the subcutaneous inoculation of 1 cc. of virus in 1:100,000 dilution, and that one positive subclinical case of mosquito transmission was observed.

Following a diagnostic inoculation the temperature should be taken twice daily. In positive cases there is a sharp rise in from eight to fourteen days, and there may be a slightly bloody nasal discharge. Lühr observed absence of fever following experimental inoculations that resulted in death from infectious anemia, but this experience is infrequent.

Various special diagnostic tests have been advocated recently. The *mercuric chloride test* consists in the addition of 1 cc. of serum to various bichloride of mercury solutions. Flocculation at 1:100,000 is regarded as positive. Vallée has reported reactions in 14 of 16 anemia cases, in 13 of 58 normal horses, and in 5 cases of strangles. Ikeda, in Japan, obtained 1 reaction from 32 infected horses. Mohler[15] has also reported the test to be unreliable.

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Opperman[16] reports that doves are susceptible to the virus of swamp

fever, and that the serum of an infected dove will agglutinate the red blood cells of guinea pigs. This opinion has gained little support.

Control.—Scott[10] states that all diseased and infected horses should be killed. While this is true from a sanitary standpoint, the detection of all carriers presents many difficulties. In sections where the disease is endemic on certain farms, restriction of all horses to this farm until there has been no evidence of the disease for at least two years is indicated. Since water from stagnant pools may contain the virus, as demonstrated by Fulton,[4] improved water sources are desirable. In swamp fever districts swampy pastures should be drained, or their use for horses discontinued. The temperatures of suspected horses should be taken daily. Scott states that if the period of temperature readings is between July 15 and December 1, taking the reading for three months will usually disclose some evidence of the disease; if the period lies between December 1 and July 15, the temperature should be taken for at least five months. Severe work will usually induce onsets of the fever in short periods. All attempts to induce artificial immunity have failed.

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INFECTIOUS BRONCHITIS OF EQUINES

(Contagious Cough of Horses; Enzootic Laryngotracheitis; Epizootic Catarrh; Grippe; Skalma)

Definition.—Enzootic cough of equines is a highly contagious bronchitis and peribronchitis characterized by a dry harsh cough and caused by a filterable virus.

This disease has been chiefly reported in European literature under various names. It was described by Dieckerhoff[1] as *Skalma*, and by Hutyra and Marek[2] as epizootic laryngotracheitis of horses. It has been regarded as a special form of influenza, and Finzi is quoted as having shown experimentally that it is etiologically identical with equine influenza. In a report on epizootic contagious catarrh of the respiratory organs of equines, Theiler[3] has explained the confusion which exists in the classification of this group of affections. Two definite specific infectious diseases have been recognized, namely, equine influenza and equine pneumonia, but many have believed with Finzi that the Skalma of Dieckerhoff was only a mild form of influenza. That it constitutes an independent virus disease has been reported by Waldmann and Köbe.

Etiology.—Chief interest in this disease has centered in training stables of purebred horses where its rapid spread has curtailed activities. But it also occurs generally in all breeds, and there is little doubt of its frequent prevalence among horses throughout the world. Recovery always occurs in uncomplicated attacks, but secondary infections may lead to pneumonia. The diseased animal is the chief source of infection and the degree of spread by intermediate carriers is not known.

In the experiments conducted by Waldmann and Köbe,[4] transmission occurred in three days in a yearling colt placed between two stalls occupied by sick horses. Transmission also occurred when germ-free filtrates from lung tissue were placed in the throat. Bacteriological examinations of slaughtered, infected experimental animals were negative. In prolonged attacks, however, a secondary streptococcic pneumonia sometimes developed; apparently the streptococci were identical with those found by Schütz in contagious equine pneumonia (*Brustseuche*). Following infection with germ-free filtrate, secondary streptococcic infection was observed only once. While the virus is the only contagious

element, natural infection may implant both the virus and the streptococci.

Experimental inoculation was positive in bovines and swine.

Symptoms.—Following natural infection or artificial inoculation the first reaction is a rhinitis and a conjunctivitis. The nasal mucosa is red. The larynx and first tracheal ring are sensitive to pressure. These prodromal signs are followed in from one to four days with a cough; this is forceful, dry, and apparently painful. There is a transient fever; this occurs with the prodromal signs or at the onset of the cough. Under usual observations of practice the fever may not be observed, and it may be missed when the temperature is taken only once daily. It is thought that the fever is concurrent with the passage of the virus into the circulation. At the time of the fever or the onset of the cough the lymph glands of the throat are swollen and painful. These inflammatory signs, like those of the mucosa, persist for only three or four days. There may be dullness, inappetence, and shivering during the febrile stage. The attitude and appetite are normal when the fever recedes, regardless of the cough. During the further course of the disease the cough is the only symptom. If the patient is not worked the cough recedes and in one to two more weeks recovery is complete. Apparently one attack confers immunity.

Secondary infection begins with a rise of temperature, persistent cough, and loss in condition. The nasal discharge is yellowish or yellowish green mucus that may be discharged en masse with the cough. The conjunctiva is red, sometimes yellowish. The lymph glands of the larynx are swollen, but never form abscesses. Ausculation of the lungs gives sharp vesicular breathing and occasional slight râles. Pleuritis is infrequent.

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EQUINE INFLUENZA

(Catarrhal Fever, Pinkeye, Epizootic Cellulitis)

Definition.—An acute highly contagious general infectious disease of equines. Usually the infection localizes in the respiratory mucous membrane, less often in the mucosa of the digestive tract, and in the subcutis. Complications are frequent. The cause is a filtrable virus with which secondary infection is often associated. An epizootic swept America in 1872-73, when horse transportation in large cities was paralyzed for weeks. During World War I it was constantly present among newly purchased horses and mules of all armies. In stockyards, camps, remounts, wherever susceptible animals are assembled, the disease appears. It resembles influenza in man in the sudden onset, high fever, variable type, rapid spread, and numerous severe complications. Certain physicians have even fallen into the error of believing that the two are identical.

Etiology.—Little is known of the nature and distribution of the virus of influenza. Until recently it has been confused with that of contagious equine pneumonia (*Brustseuche*). Artificial transmission by means of blood, filtered blood serum, and semen, has been reported by various investigators (Gaffky,[2] and Bergman[1]). The period of incubation is from two to six days following subcutaneous injection. The virus is present in the blood and other body fluids of the sick, where it may remain an indefinite time. Basset reported finding it in the blood months after recovery, while Bergman reported finding it in the semen of a stallion six and one-half years after recovery. It is improbable that recovered animals regularly transmit the disease.

The virus is spread readily by the sick, and objects with which they come in contact—mangers, water pails and troughs, cars, utensils and grooms. The disease spreads so fast that after it once appears in a susceptible group there is slight chance for prevention. We have no definite knowledge of the length of time the virus remains alive in infected yards and stables, but apparently it soon dies out when susceptible animals are withheld. The more serious epidemics occur in remounts, camps, and yards where arrivals of green and susceptible animals are frequent, and where the virus may be kept abundant and virulent for a long time. Among farm animals the disease is sporadic or slightly endemic, usually mild, and is nearly always introduced by a horse that has recently passed through a stockyard. One attack confers permanent immunity. While the presence of the filtrable virus is essential to the development of influenza, chilling and fatigue may overcome natural resistance and cause the attack.

Morbid Anatomy.—Since death results usually from pneumonia this is the usual finding on autopsy. The lungs show hyperemia, edema, and hemorrhagic bronchopneumonia. The mucosa of the digestive tract may be hyperemic, hemorrhagic and swollen. Gelatinous infiltration is often present around the pharynx. The large body glands and lymph glands have undergone changes like those of septicemia—degeneration of the heart, kidney, liver and muscles. Other changes are subcutaneous and subserous hemorrhages, swelling of the lymph glands, and serous transudation into the body cavities. Death may also result from circulatory failure or enteritis.

Symptoms.—The period of incubation is from five to ten days. The disease sets in suddenly with chills, fast breathing, and complete loss of appetite. In some, prostration is marked by drooping of the ears and resting the head on the manger. Others are restless and change position often, when one may hear creaking sounds in the joints. A fever of 103° to 106°F. continues for about three days. The conjunctival mucosae are congested and often jaundiced.

The chief localization being in the upper *respiratory tract*, a harsh cough is present from the beginning. The nasal mucous membranes are reddened and at first there is a bilateral serous discharge which may become purulent; in some there is little or no nasal discharge. Cough and induced cough are present with few exceptions. Symptoms of pharyngitis in the form of a painful suppressed cough, swelling of the throat, and abundant purulent nasal discharge are met with occasionally. Examination of the chest reveals an increased vesicular murmur, and the breathing shows a slight expiratory lift.

Influenza pneumonia is a serious and often fatal complication. This may appear at any time after the first two or three days, especially in animals that are exposed and given poor care. It may be associated with fibrinous pleuritis.

The *digestive symptoms* are a depressed peristalsis and slight constipation followed occasionally by a slight fetid diarrhea. In certain years one meets with an intestinal type in the form of a highly fatal gastroenteritis; this should not be confused with a terminal diarrhea.

Epizootic cellulitis, or "pinkeye," is generally considered to be a form of influenza marked by edema of the subcutis of the limbs, ventral parts of the body, and often the eyelids. The legs may be swollen to a uniform diameter their entire length. The eyelids may be closed by an edematous swelling, while there may be conjunctivitis, turbidity of the cornea, and even lesions within the eyeball—iritis, cataract. Pregnant

mares abort. Stallions are affected with orchitis. According to Williams,[3] and others (Luckey[4]) epizootic cellulitis is an independent disease.

Other irregular forms are laminitis, swelling of the tendon sheaths and joints, urticaria, nephritis, paralysis from lesions in the brain or spinal cord, and muscle cramps. In uncomplicated attacks the fever usually recedes on the third day and recovery from the acute symptoms occurs in one to two weeks. The mortality is estimated at from 0.1 to 1.0 per cent, though it may be as high as 4 per cent, and in the intestinal form much higher.

Diagnosis.—Influenza may be confused with pharyngitis and other affections of the upper respiratory system. The high fever, prostration, and contagion identify it clearly from mild local catarrhs. The pneumonic form is not readily differentiated from contagious pneumonia when they exist together as in a camp or stock yard. If the individuals are carefully watched it will be observed that primary pneumonia appears earlier in the attack. A diagnosis of influenza is often made carelessly without regard for its characteristic features.

Treatment.—In typical uncomplicated attacks, absolute rest and protection against cold, moisture, and drafts are of first importance. Provide fresh water often. In severe localization in the pharynx, steam inhalation gives relief. Strychnine sulfate (gr. i) thrice daily combats prostration. Expectorants of ammonium chloride and ammonium carbonate with belladonna to relieve cough are indicated—see coryza. Constipation may be corrected with sodium sulfate 1 to 2 ounces (30-60 Gm.) daily in an electuary or liquid petrolatum 250 cc. or a diet of carrots. Aloin and other active cathartics are contra-indicated. Circulatory weakness is treated, as in pneumonia, with sodiobenzoate of caffein 2 to 4 drams (8-16 Gm.) or camphorated oil 4 to 8 ounces (120-240 cc.). Pneumonia and other complications should receive appropriate treatment. Where many animals are together, constant watching is desirable in order to remove sick animals promptly. Each sick animal should be tied separately to avoid disturbance from well and convalescent individuals. Food and water should be brought to the sick.

The use of anti-hemorrhagic septicemia serum, prepared from *Pasteurella equi*, in the treatment of 100 cases of "*shipping sickness of horses*" under sales-stable conditions has been reported by Marshall and Lee.[5] The dosage was 100 cc. intravenously. Many of their cases received only one treatment, and no other treatment was prescribed. In conclusion they report as follows: "The writers have been familiar with sales-stable conditions for years and have never known of a more satis-

factory treatment than is shown in the report of these 100 cases." In the same bulletin, they refer to apparent benefit from the use of hemorrhagic septicemia aggressin (5 cc.) as an immunizing agent. Antihemorrhagic septicemia serum (200-250 cc. monthly) has been used rather extensively as a prophylactic against influenza in race horses, and in young animals previous to shipment. In the treatment, apparent benefit has resulted from blood transfusion, using blood from an acclimated horse, or from one that has recovered from an attack of influenza within a year.

In the treatment of pneumonia secondary to, or combined with, strangles and influenza in recently shipped army remount horses, sulfanilamide was found by Seymour and Stevenson[6] to be more useful than neoarsphenamine. The sulfanilamide was given over a period of 4 days in doses of 90 Gm. daily the first and second days, and 60 Gm. daily the third and fourth days. To each dose of sulfanilamide 30 Gm. of sodium bicarbonate was added to reduce the acidity and overcome toxicity. This mixture was suspended in 2 liters of water and given through a stomach tube. When the stomach tube could not be passed because of pharyngitis the patient received 500 cc. of citrated whole blood once or twice daily. In the treatment of uncomplicated attacks of influenza Seymour and Stevenson[6] reported excellent results from the early administration of neoarsphenamine 3 Gm. in 200 cc. distilled water intravenously, repeated every other day until at least three or four doses have been given. Antibiotics are indicated.

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CONTAGIOUS EQUINE PNEUMONIA

(Lobar Pneumonia; Croupous Pneumonia; Brustseuche; Pasteurellosis)

Definition.—Equine lobar pneumonia is an acute general infectious disease having a definite course and a typical high fever lasting about one week. The lung lesions, unlike lobar pneumonia in man, are variable in character throughout the hepatized lobe.[1] It occurs chiefly where many horses are assembled, and where new ones are frequently added. Thus it is common in remounts, lumber camps, in transit (ships, trains), stock yards, and sales-stables. During the period of the World War losses from this disease were heavy.

Etiology.—Many have sought for a specific cause of equine pneumonia. Gaffky and Lühr[2] conducted what perhaps was the most extensive investigation of disease in the horse that has ever been undertaken. For a period of about ten years many horses in the German army were utilized for their purpose. Finally, they were able to transmit the disease artificially by smearing on the oral and nasal mucosa of susceptible animals a fibrinous bacteria-free exudate obtained from the smaller bronchi of horses slaughtered after a period of sickness of from three to four days. The incubation period in these experimental cases was from eighteen to forty-two days. It is not infrequent for a naturally exposed animal to sicken in less time than observed in Gaffky's experiments, which suggests the possibility of another virus. In 1887 Schütz attributed the cause of equine pneumonia to *Streptococcus pyogenes equi*, and in 1897 Lignières attributed it to *Pasteurella equi;* these are now regarded as secondary invaders. Following transportation of green horses the mortality from pneumonia is often high, especially when they pass through stock yards. While pneumonia is relatively infrequent and mild among small groups of mature farm horses, the assembling of such animals in large numbers is often followed by a fatal outbreak, regardless of the season. Pneumonia may develop within twenty-four to forty-eight hours after severe chilling or fatigue. Hutyra calls this a *genuine* croupous pneumonia as distinguished from the contagious form. Until our knowledge of the bacterial cause of pneumonia is more complete each case should be regarded as contagious, even when atypical or sporadic.

Morbid Anatomy.—The postmortem findings are variable; they depend on the virulence of the infection, the stage at which death occurred, and the character of the secondary changes. When death occurs early, during the stage of pulmonary congestion, this is the chief lesion. As a rule death occurs after the pneumonia is well developed. In some the

pneumonic lesion is slight in the form of a lobular pneumonia, or it may develop as a lobar pneumonia with extensive consolidation in one or both lungs. Usually the consolidation is confined to the ventral lobes and the overlying part is emphysematous. In extensive advanced consolidation the cut surface presents a firm reddish surface containing grayish areas. Microscopic section of the red portion shows hyperemia with exudation of fibrin or leucocytes into the alveoli. Microscopic section of the gray portion shows marked infiltration with leucocytes; extensive areas may be found where the tissues have lost their structure and are bordering on necrosis. The visceral pleura may be inflamed and thickened, and over the parietal pleura one sometimes finds thick masses of fibrinous exudate. The thorax often contains an abundant reddish serum. The remaining postmortem changes are like those which characterize septicemia, namely, degeneration of the kidneys, liver, and spleen, with various degrees of congestion or inflammation of the intestines.

Symptoms.—Among susceptible horses placed with the sick, Gaffky observed an incubation period of twenty-two to forty-four days. Others have reported a period of one to three weeks in animals exposed to overexertion and fatigue. Because of lack of knowledge of the time of infection the incubation period under natural conditions is difficult to determine. It is longer than in most acute general infections, and more time is required for the epidemic to assume dangerous proportions. Transmission seems to depend largely on direct contact. As a rule, the disease sets in abruptly with a chill, a temperature of 104° to 106°F., a pulse from 50 to 100, respirations from 20 to 60 and somewhat abdominal in type. In milder forms there are sometimes slight catarrhal symptoms for two or three days. Occasionally a brownish or yellowish slight exudate appears at the nostrils on the first or second day; this is pathognomonic of pneumonia. The darker exudate represents a capillary hemorrhage of active pulmonary congestion; the lemon-yellow discharge is a part of the fibrinous exudate from the bronchi. At no stage of a typical lobar pneumonia is there more than a slight nasal discharge, and in most cases there is no discharge whatever. Cough is always present and it may be easily induced by pinching the larynx; it is low, moist, and often painful and suppressed.

On *auscultation* over the lungs one may hear a variety of râles, depending on the stage of the disease, the extent of the lesions, and the rate of consolidation. Râles vary from time to time according to the distribution and consistency of the bronchial exudate. At first one hears only a harsh vesicular murmur (congestive stage). In from twelve to twenty-

four hours after the onset, one may hear crepitant râles. These are high-pitched inspiratory sounds believed to be produced by the separation of the sticky surfaces of the finer bronchi and alveoli. As the exudation increases a variety of musical and snoring râles may be heard until resolution sets in, when the sounds gradually diminish to the rough, and finally to the normal vesicular murmur. When consolidation is extensive and complete, there may be complete absence of sound over the affected part. When consolidation is extensive and incomplete, the bronchi remaining open, one may hear bronchial breathing. This is a loud stenotic, blowing, rushing, or tubular sound that presents an intensity greater than in any other lung affection. It is the transmission of the laryngeal and trachael sounds through the bronchi and consolidated lung tissue. Pulmonary disease may also be recognized by hearing in one lung a sound that is absent from the other.

Percussion over the chest may induce either pain or cough, symptoms that are never met with under normal conditions. In active engorgement, percussion reveals a tympanitic sound; in extensive hepatization, a dull sound. In heavily muscled draft horses percussion may be of little value. Bronchophony gives an increased sound over the affected lung; it is recognized by auscultation over the chest while an assistant percusses the trachea. The results of a physical examination of the chest vary widely according to the nature of the case, the stage of the disease, the type of animal, and the skill of the examiner.

Toxic circulatory weakness is frequent and is recognized by general weakness, sweating, a small, soft, arrhythmic pulse, increased heart impulse, and distended peripheral veins. The *fever*, in uncomplicated types, recedes rapidly or slowly at the end of about a week, and this is followed by gradual recovery.

Complications are rather frequent and severe in large groups kept under unfavorable conditions, as in stock yards and remounts. *Heart weakness*, appearing about the fifth day, is regarded as most frequent. Under present-day interpretation, this is not a complication but a toxic vasomotor paralysis characteristic of the disease.

Pulmonary gangrene is not infrequent. While it may appear at any time, it usually develops on the seventh to the tenth day. A sudden chill in the course of the attack is suggestive. A sweetish fetid breath is pathognomonic, and this odor may permeate the entire stable. Recovery from gangrene is extremely rare.

Pleuritis.—In all pneumonia there is some degree of pleuritis. But from the middle to the latter part of the course it may become extensive. In one of the writer's cases pleuritis was the chief lesion. It is marked

by labored expiratory breathing, continued high pulse and fever, and an area of distinct dullness over the lower third of the chest. The area of dullness is marked above by a straight horizontal line, the percussion sound passing abruptly and distinctly from dullness to resonance.

Intense *icterus* indicates dissolution of the blood—hemoglobinemia. *Purpura* is a common sequella in certain severe outbreaks. Other complications are urticaria, arthritis, tendovaginitis, laminitis, encephalitis, and meningitis.

The Course.—In a typical uncomplicated case the fever recedes after a week to ten days and progressive improvement to complete recovery follows. In many cases, however, the course is changed by one or more of the numerous complications or sequellae, as in influenza. Such complications develop when the animal is returned to work before convalescence is complete or is exposed to atmospheric extremes or to the fatigue of transportation. But in addition to the complications which may occur under unfavorable conditions, these also are frequent in animals that receive excellent care and treatment.

Prognosis.—When many animals are together the mortality may be as high as 20 per cent under unfavorable conditions. Epidemics are often deceiving. At first there may be only a few sick horses and a low mortality. But in the course of one to three months it may become widespread and highly fatal. Among scattered farm horses the death rate is low. Unfavorable signs are circulatory weakness with a pulse of 80 or more and poor in quality; extensive bilateral pneumonia or pleuritis; fast breathing; blood-stained or "prune juice" nasal discharge; remittent fever, or one that persists for more than a week; diarrhea; and rapid refilling of the chest cavity with fluid after it has been aspirated.

Treatment.—The treatment and general care of pneumonia are described under bronchopneumonia, p. 46, and pleuritis, p. 78.

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SWINE INFLUENZA

(Hog Flu)

Definition.—Swine influenza is a specific and highly contagious disease induced by combined infection with a filterable virus and *Hemophilus influenzae suis*. It occurs chiefly in the fall and early winter in

the Middle West, and is characterized by fever, bronchitis, and bronchopneumonia with pulmonary edema.

Etiology.—Although swine influenza had previously been recognized in Iowa and other states, its wide prevalence in 1918 attracted special attention, when it was named "hog flu" because of its apparent resemblance to the prevailing serious pandemic of human influenza. It recurs each fall, mostly in November and December, in the Middle West. Exposure to cold weather is regarded as an important predisposing cause. Swine influenza following exposure and shipment to the Philadelphia Stock Yards, and associated with *Pasteurella suiseptica* infection, has been described by Scott.[1]

Bacteriology.—In 1931 Lewis and Shope[2] obtained from the respiratory tracts of field cases of swine influenza cultures of a hemophylic bacillus which they named *Hemophilus influenzae suis*. Efforts to induce symptoms by intranasal injections of pure cultures were negative. Shortly thereafter Shope[3] demonstrated a virus in Berkefeld filtrates which induced a mild illness in pigs when introduced intranasally. When this pure culture of virus was mixed with a pure culture of *H. influenzae suis* and then inoculated intranasally into swine, a disease identical clinically and pathologically with swine influenza was induced. Shope[3] concluded that neither infection, alone, is capable of producing the disease. In a study of immunity to swine influenza, Shope[4] demonstrated that only the filterable virus possesses immunizing properties, and that "intramuscularly administered swine influenza virus was incapable of inducing illness but did render hogs immune to swine influenza." This suggested that "a specific relationship, as regards infectivity, exists between the swine influenza virus and the tissues of the respiratory tract."

Additional investigation by Shope has revealed where the virus exists during the eight or nine months elapsing between the yearly epidemics when the swine population is free of influenza. *H. influenzae suis* can persist indefinitely in the upper respiratory tract of recovered swine, but similar persistence of the virus cannot be demonstrated. Shope[5] has now demonstrated that "lungworm larvae from pigs with swine influenza harbor swine influenza virus throughout their development both in their intermediate host, the earthworm, and their definitive host, the swine." The virus reaches the respiratory tract in the lungworm embryos and becomes infective under sufficient stimulus; such stimulus was supplied by Shope by intramuscular injection of *H. influenzae suis*, as well as by a single intrapleural injection of calcium chloride solution. Cultivation of the pneumotrophic viruses of pig influenza and swine influenza on

allanto-chorionic membrane of the chick embryo has been reported by Köbe and Fertig.[6] After such cultivation pig influenza virus lost its infectivity for the white mouse, while the swine influenza virus retained it.

Morbid Anatomy.—The postmortem changes are confined chiefly to the respiratory tract. In cases which have terminated in natural death the pleural cavities contain reddish serum, and there may be an adhesive fibrinous pleuritis. The cervical lymph glands are swollen and congested. The trachea and bronchi contain a mucous exudate and their mucous membranes are congested; in the smaller bronchi the exudate may be tinged with blood. In the lungs there is a consolidation of the anterior ventral lobes, while the dorsal and posterior parts are distended, congested, and edematous. On cut section the interlobular connective tissue is found to be thickened and the cut surface exudes a frothy bloody fluid. The mucosa of the upper air passages is congested and inflamed.

Symptoms.—The incubation period is from two to seven days. The onset is sudden and the incidence in infected herds may be nearly 100 per cent. General symptoms are severe in the form of marked prostration, anorexia, and a high fever (104° to 107°F.). Owing to muscular soreness the hog may squeal from pain if handled. The respiratory symptoms are labored abdominal breathing, cough, thumps, and a mucous discharge from the eyes and nose. The course is from four to six days and in uncomplicated cases recovery is prompt. The mortality is from 1 to 4 per cent, though in seasons when the epidemic is severe the mortality may reach 10 per cent. Recovered animals were found by Shope to be immune to artificial inoculation, yet McBride states that the same herd may suffer from two or even three attacks in the same season. Often the outbreak is in the form of a mild nonfatal bronchitis which causes a loss in condition.

Treatment.—It is probable that pig influenza (Ferkelgrippe) and swine influenza are identical, and that the brief descriptions of the disease in America have been based on observations on older swine. Unrecorded reports of high mortality in young pigs following return of others from public exhibits support this view. The most effective herd treatment is to provide warm, dry, clean pens, and to feed sparingly. Sulfamethazine in mash at the rate of 1½ grains per pound body weight the first day and 1 grain per pound on the second and third days, as well as 25 per cent solution of sodium sulfamethazine intraperitoneally at the rate of 1 grain per pound of body weight per day on three successive days, was followed by immediate improvement in an outbreak of pneumonia in swine.[7]

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PIG INFLUENZA

(Ferkelgrippe)

Waldmann[1] has described a form of influenza in pigs in Germany which is caused by a pneumotrophic virus closely related to that of swine influenza; he suggests that it may be a weaker form of the swine influenza virus. Losses from this disease are reported to be higher in Germany than from swine erysipelas and hog cholera combined. It spreads rapidly through the herd, affecting pigs up to 6 weeks of age, and causing a mortality of about 50 per cent. The symptoms are conjunctivitis, apathy, inappetence, and cough; chronic cases remain stunted. In older swine the symptoms are mild and the mortality is low. On postmortem examination bronchopneumonia is found. The *virus* within the body is found only in the lungs and adjacent lymph glands; rarely and briefly it is present in the blood. Outside the body it soon succumbs. The mode of infection is only by direct contact from animal to animal, or by droplet infection when the animal coughs. There are no intermediate carriers.

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EQUINE ENCEPHALOMYELITIS

(Virus Encephalitis; Sleeping Sickness)

Definition.—Equine encephalomyelitis is an infectious disease caused by a neurotrophic virus, and characterized by derangement of conscious-

ness, motor irritation, paralysis, and a high mortality. In the usual form there are no postmortem changes visible to the naked eye, but perivascular round cell infiltrations are present in the encephalon and to some extent in the spinal cord. In the United States two types of virus have been recognized: a highly virulent *eastern* and a less virulent *western* type. Natural infection has been observed in this country chiefly in horses, but in 1938 encephalomyelitis in children due to equine encephalomyelitis virus of both eastern and western types was discovered (U.S.B.A.I.),[1] and natural epidemics in pigeons and pheasants were likewise observed.

Etiology.—Equine encephalomyelitis was first described in Europe where it has been prevalent in southern Germany for many years. The European form of endemic encephalomyelitis (Borna disease) has not been recognized elsewhere. While Borna disease occurs chiefly in horses, it has been described by Miessner[2] and others in sheep and cattle. In 1927 Zwick[3] reported the cause to be a filterable virus. In contrast with the form observed in the United States, the period of incubation in experimental animals is longer, the American and European viruses do not cross-immunize, the seasonal prevalence is wider, and the nuclei of certain nerve cells contain "inclusion bodies" which usually have not been observed in the brain tissue of affected horses in the United States. In the Argentine an enzootic of encephalomyelitis caused by the western type of virus has been described. In Venezuela the disease has been caused by a type of virus unlike that observed in any other country; thus, there are at least four types of virus. All three strains known to be present in the Western Hemisphere are capable of producing fatal encephalitis in man, and Rivers[4] writes that in 1941 there were 3000 cases and 200 deaths in man from the western type in the Dakotas, Nebraska, Montana, and Manitoba.

In 1912 an extensive outbreak of encephalomyelitis occurred in Kansas and adjacent states where it was widely prevalent from early in August until the coming of frost in September and October. In this area approximately 35,000 horses died. The symptoms were characteristic of encephalitis—deranged consciousness, motor irritation, and paralysis. On autopsy no macroscopic changes were found. Microscopic examination of the brain tissue revealed perivascular cellular infiltration, but no intranuclear inclusions as observed in Borna disease. The epidemiology, symptoms, and lesions indicate that it was an infectious disease similar in character to Borna disease. It was described by Udall[5] as an encephalitis, probably caused by infection. At the time of the outbreak it was generally regarded as a form of forage poisoning, but the symptoms and wide distribution were unlike those of any form of forage

poisoning that has ever been described. In 1931 Meyer[6] reported that in the years 1915-1920 an approximate mortality of 3000 horses has been estimated for the five western states, California, Colorado, Oregon, Nevada, and Montana, and that in recent years maladies of horses with symptoms of encephalitis have been seen in Kansas. The theory that this disease is a form of forage poisoning, or that changes in the food or water are in any way associated with the cause, was disproved in 1931 through the discovery by Meyer[6] and associates of a filterable virus in the brains of horses affected with enzootic encephalitis, in California. In 1933 this disease assumed increasing importance because of its appearance in the East in the form of a destructive outbreak among horses along the Atlantic Coast in Maryland, Delaware, and Virginia.

Since the epidemic of encephalomyelitis among equines in California in 1931, the disease has spread rapidly over the western and midwestern states and western Canada, reaching its height in 1938 when 184,662 cases were reported during the summer and fall. Since 1938 the numbers have varied from approximately 3000 to 45,000 annually with an average of about 14,000. In general the eastern type occurs only in the states along the Atlantic, and the western type only in the states west of the Appalachian Mountains; yet the western type has been identified in Alabama, while the eastern type has been found in Missouri and in Texas several hundred miles inland from the Gulf. In 1951 only 762 cases were recorded.

The seasonal occurrence in the United States has been from June to November or the appearance of frost. In Europe, Borna disease is most prevalent from March to August, reaching its height in midsummer, but it may occur during any month. There is no age immunity. Importance has been attached to the influence of moisture in the atmosphere and in low-lying districts as a contributory cause, yet the disease in America has been widely prevalent in exceptionally dry areas, such as the San Joaquin Valley, Nevada, and western Kansas. The epidemiology is similar to that of infantile paralysis in man; possibly infected animals acquire immunity without showing recognizable symptoms. In Kansas the number of sick animals in a herd averaged about 20 per cent. The first animal to sicken may not have been off the farm for weeks, and not within miles of a recognized case. Several days may elapse between attacks. The disease is limited almost entirely to farm horses. Because of its widespread distribution it is met with in animals fed a wide variety of foods and given drinking water from many different sources.

The Virus.—In 1927 Zwick[3] reported that Borna disease is caused by a filterable virus located in the brain tissue, the salivary glands and

saliva, and the nasal mucosa. With an emulsion of brain tissue from a horse dead of Borna disease, he produced the disease in rabbits experimentally by feeding and by intracranial or sciatic nerve inoculation. The guinea pig, rat, sheep, and fowl were susceptible. Inoculation of cattle was negative. Following intracranial inoculation the period of incubation was seven weeks in the horse and three to four weeks in the rabbit. The disease also spread among rabbits by cohabitation. In the United States no virus of encephalitis had been found, previous to the investigation made by Meyer and associates,[6] in 1931. Their studies proved conclusively the presence of infective virus. This was a filterable virus obtained from the brain and was infective to horses, monkeys, rabbits, guinea pigs, rats, and mice, by subdural or intracerebral injections of brain suspensions. Intracerebral and intranasal instillations of brain emulsions caused deaths in guinea pigs in four to six days. Rabbits were readily immunized. They believed the infection to be identical with that previously active on the American Continent, but not with Borna disease. Many mammals and birds are susceptible to experimental inoculation.

In the tissues of affected horses the virus is most commonly found in the brain. It has been found in the blood stream during the febrile stage, on the nasal mucosa of the sick, in the salivary glands and in the saliva, and recovered animals may be carriers. Its presence, however, is not constant; it may be impossible to demonstrate the virus in the body at the time of death, and it cannot be found a few hours after death. The virus is best recovered from animals slaughtered in an advanced stage of the disease.

In 1933 transplantation of the virus to laboratory animals and horses by bites of experimentally infected mosquitoes (*Aedis aegypti*) was demonstrated by Kelser.[7] Subsequent work on transmission by insects led to the view that mosquitoes are chiefly responsible for natural infection in equines. In 1938 Tyzzer[8] and associates discovered that equine encephalomyelitis virus occurs naturally in ring-necked pheasants. Specimens were received from the field in Connecticut where the disease was highly prevalent and where many dead wild birds were found after a hurricane. The infective brain tissue transmitted the disease to Swiss mice in forty-eight hours after intracerebral and in three to four days after intraperitoneal injections; the strain was carried through 10 passages in experimental inoculations. In their report Tyzzer and associates question "whether the horse or other domestic animals are responsible for the wide distribution of the virus." They suggest that the virus is distributed by migratory birds. This suggestion has been supported by the discoveries of Reeves and Harmon,[9] who con-

clude that in the Pacific Coast States the mode of transmission is through the agency of *Culex* mosquitoes which feed on birds. They carry the virus from the heavily infected domestic fowl and possibly wild birds, where it causes no disease, to both horse and man. Other biting pests, including ticks, mites, fleas, lice, flies, kissing bugs and bed bugs were tested as carriers, but only *Culex* mosquitoes were found to be infected. It is probable, however, that the disease-free "reservoir" of infection and the intermediate carrier or vector differ from one area to another. In 1940 Kitselman and Grundmann[10] demonstrated virus in the kissing bug, *Triatoma sanguisuga*, the first isolation of virus from a naturally infected insect, and in 1945 Sulkin[11] reported finding virus in chicken mites on a farm in Texas. The susceptibility of birds probably accounts for the massive concentration of encephalomyelitis virus in chick embryos. During the 1938 epidemic in southeastern Massachusetts there were many losses of pigeons which ceased with the beginning of cold weather. From a dead pigeon that had spontaneously contracted the disease Fothergill and Dingle[12] recovered the virus of the eastern type of encephalomyelitis.

In 1933, TenBroeck and Merrill[13] observed that the eastern virus was more virulent than the western, and that sera from eastern horses that had recovered from the disease neutralized all strains of eastern

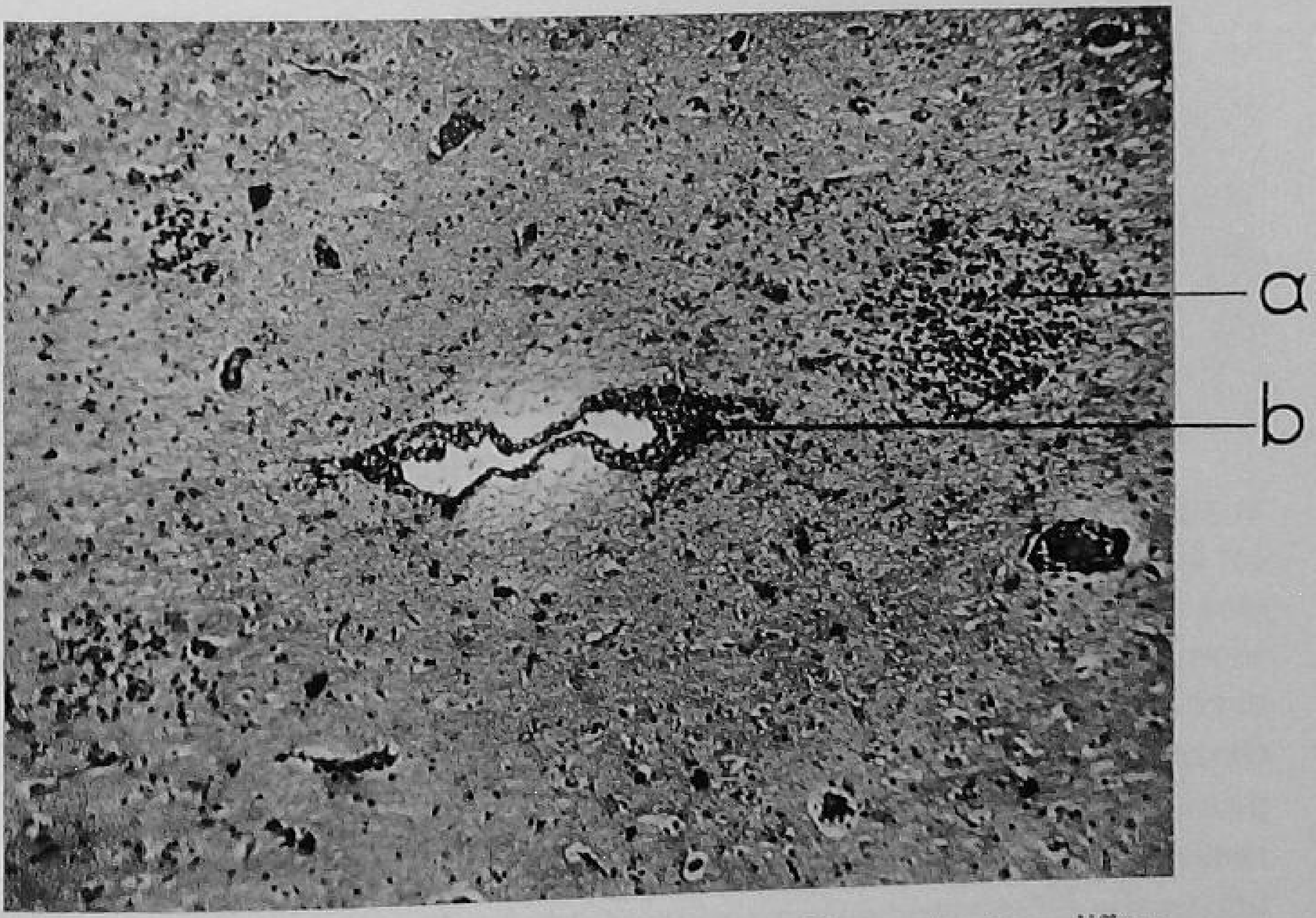

Fig. 75.—Enzootic encephalomyelitis. Section from olfactory tract: a, diffuse interstitial infiltration; b, perivascular infiltration. × 90.

virus, but did not neutralize strains of western virus; there is, therefore, no cross immunity.

Mode of Infection.—Since it has been shown experimentally that the disease can be transmitted by insects, such as mosquitoes, it is commonly believed that they are the chief source of infection.

While it is commonly stated that recovered animals are immune, it was observed in North Dakota that 255 horses that had the disease in 1935 and recovered were again infected in 1937.

Morbid Anatomy.—No gross changes are present. Joest,[14] working on Borna disease, found the lesions chiefly in the encephalon, to a less extent in the medulla. They consist mainly in a lymphocytic perivascular infiltration in the olfactory bulb and the olfactory tract, to a less extent in the nucleus caudatus and hippocampus. It is an acute nonsuppurative disseminated inflammation of the brain. In addition, in Borna disease, intranuclear bodies are found in the ganglion cells. In the outbreak of encephalitis in Kansas in 1912, perivascular infiltration in the olfactory bulb was observed, but the intranuclear bodies found

Fig. 76.—Sleepy type.

in Borna disease were not observed. The histological changes observed by Hurst[15] in equine encephalomyelitis (eastern strain) were degeneration of the nerve cells, the appearance of nuclear inclusions resembling those of Borna disease and poliomyelitis, and perivascular cuffing with mononuclears and polymorphonuclears in varying proportions. The changes were most pronounced in the cerebral cortex; they were less pronounced in the brain stem and cervical cord. He reports that "the eastern type of encephalomyelitis is characterized histologically by an unusually diffuse and intense acute inflammation affecting most territories in the central nervous system but more particularly the grey

masses. . . . Similar changes produced in the horse by the western strain of virus are less intense and extensive."

Zwick[20] and others[21] have reported that in Borna disease the lesions are present in the spinal cord, the spinal ganglia, and the peripheral nerves, especially the brachial and sciatic.

Reporting on the lesions observed in the California outbreak, Meyer[6] writes: "No gross anatomical lesions were found at autopsy. . . . The most obvious and striking microscopic changes in the brain consisted of hemorrhages around the vessels of the olfactory bulbs and brain stem. Infiltration of the perivascular sheaths and spaces due to mononuclear and polymorphonuclear cells was variable in intensity. . . . The distribution and intensity of the inflammatory foci differ from those commonly seen in typical Borna disease."

Symptoms.—In the experimental inoculation reported by Zwick,[20] the period of incubation in the horse was seven weeks. The incubation period of the disease in California was reported to be from one to three weeks.

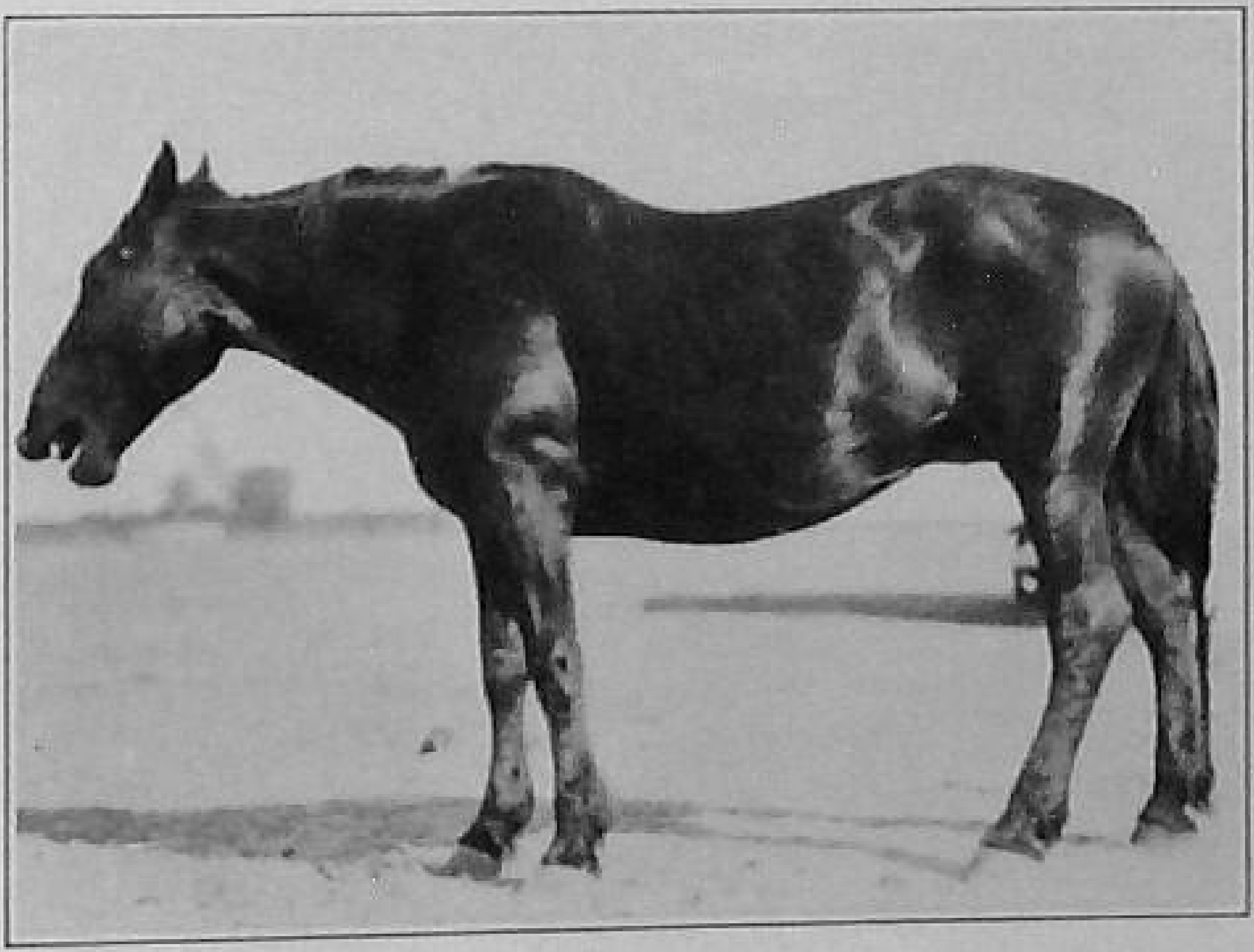

Fig. 77.—Yawning is a frequent symptom.

Nearly all of the dominant symptoms are of the nature of psychic derangement, motor irritation, and paralysis; with few exceptions all three are present. The disease may occur in so mild a form that it is hardly recognized, and such mild attacks may explain the apparent immunity of many individuals. Before the appearance of the distinctive nervous symptoms, one may note depression, and petechiae or ecchymoses of the conjunctival sclera. The attack may be sudden. In one of the author's cases, a colt that appeared to be normal became ill half an hour later and was tied between a stake and a tree. He was pressing forward strongly against the rope in a condition of great excitement.

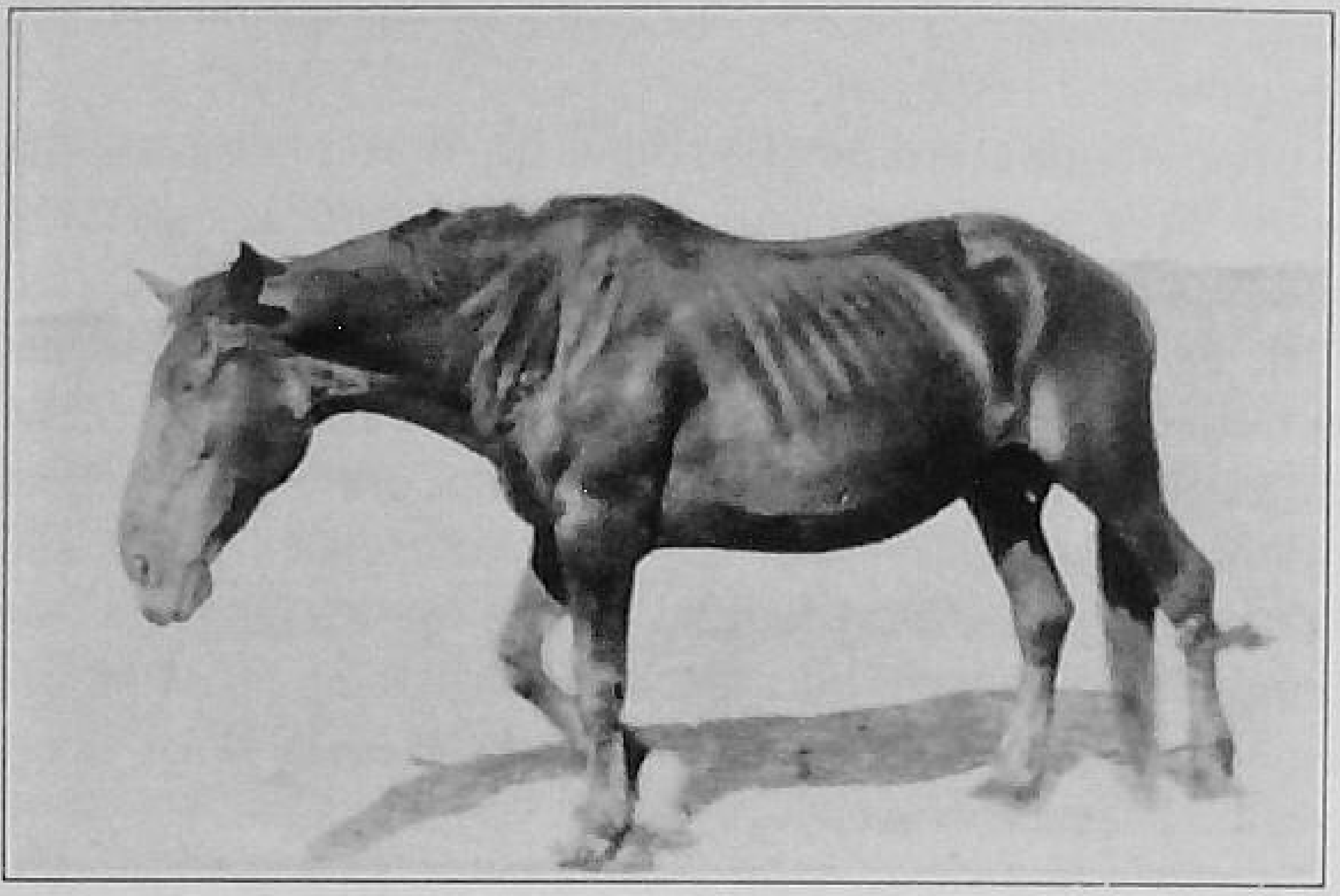

Fig. 78.—Stance when disturbed.

The skin broke out in a general sweat that dropped freely to the ground. The muscles of the body and limbs were strained, and the animal made frequent involuntary stepping movements, so that a near approach was not safe. The pulse was 90, the temperature 105° F., and the breathing fast. Chewing movements and salivation were almost constant. There

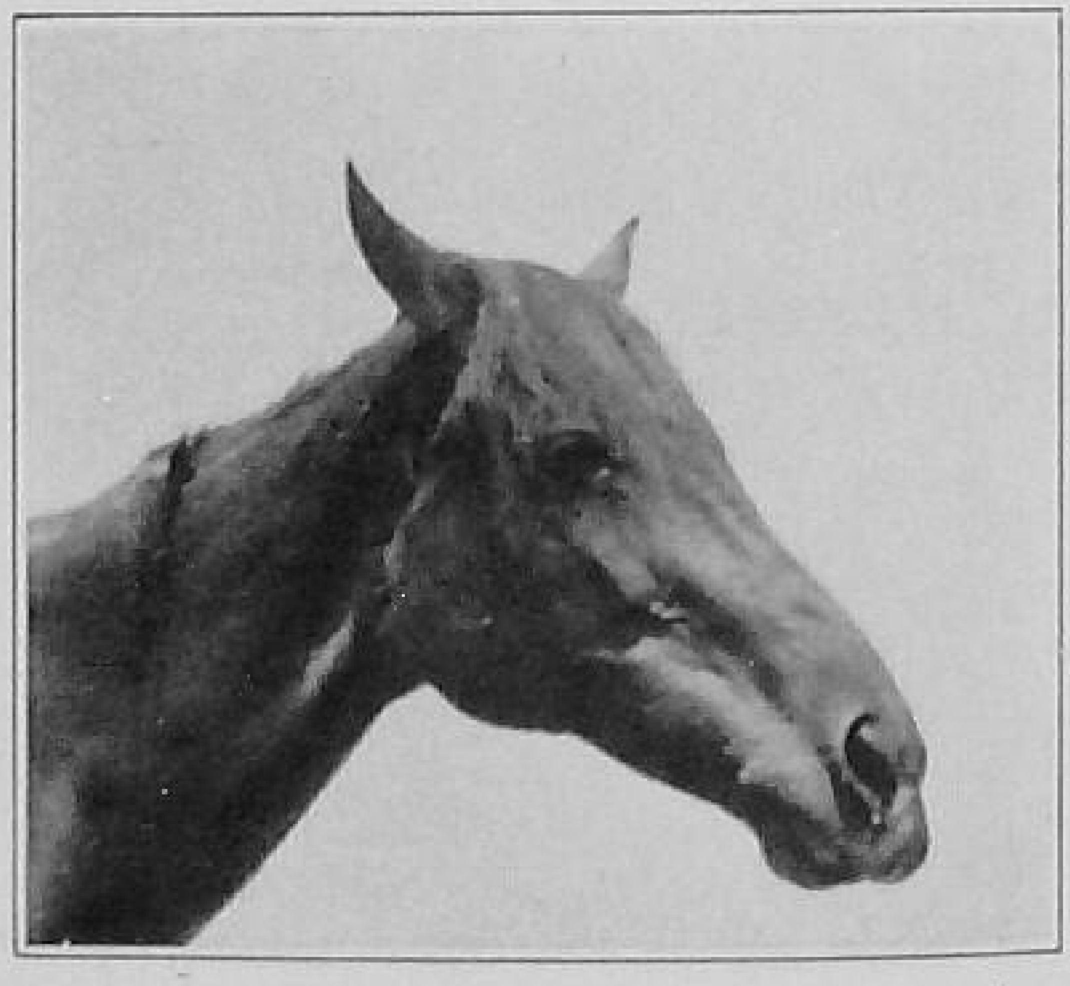

Fig. 79.—Muzzle twisted, caused by right facial paralysis.

Fig. 80.—Looseness of the lips may be one of the first symptoms noted, as in this case.

was a frequent drawing upward of the upper lip and downward of the lower lip due to clonic spasms of the corresponding muscles. There was a rapid involuntary winking of the eyes, oscillation of the eyeballs, and complete closure of the pupils. These symptoms, combined with slight involuntary urination at frequent intervals, gave a striking syndrome. After about an hour the animal became quiet and stupid.

The symptoms of *deranged consciousness* are prominent, and they indicate more than any other sign that the brain is diseased. Excitation of consciousness may cause the animal to run blindly through fences, over farm machinery, or into any object. In most cases no period of excitement is observed, and when present it is usually transient. Mental depression in some form, from dullness in the early stages to complete coma near the end, is an almost constant symptom. Horses thus affected stand constantly in one position, often with the head hanging low, and finally they go down with paralysis. When moved they walk with difficulty and may stumble and fall.

Fig. 81.—A good type of supporting frame with a strong rear crossbar and feed box high enough to keep the head level with the body. The combined use of a sling and supporting frame is desirable in severe cases; the sling alone does not suffice.

Motor irritation is commonly present, especially clonic spasms of certain groups of muscles, and involuntary forward or circling movements.

Paralysis is constant, either from the onset or shortly thereafter, finally becoming complete. Paralysis of the throat is the rule; the resultant accumulations of food and saliva give rise to a fetid odor and a nasal discharge. Special emphasis has been placed upon paralysis of the lips. Peristalsis and bowel evacuations are suppressed from the beginning and laxatives are without effect. Inhalation pneumonia is a frequent complication.

The *course* is from a few hours to a few days. At the height of the outbreak, death usually occurs in from two to four days. A few drop

dead without showing previous symptoms, and recovery may leave the animal a "dummy."

In 1912, in Kansas, the mortality was over 90 per cent. In the California outbreak in 1931, the mortality was about 50 per cent; in 1938, it was 20 per cent in the West and 90 per cent in the East.

Diagnosis.—Much confusion has resulted from failure to distinguish between paralysis alone, as seen in botulism, and paralysis associated with deranged consciousness (excitement or coma) or motor irritation (spasms and convulsions, walking in circles, pressing forward), as seen in encephalitis. Affections that may possibly be due to botulism, or unknown forms of food poisoning, are usually confined to one farm or a small area; there may be a history of consumption of spoiled food; it occurs at any season, and more often in animals not on pasture. As Meyer[6] indicates, "although the practicing veterinarian is frequently not in a position to observe and analyze the course of the symptoms, the lesions and the spread of the malady, he should, however, bear in mind that the scattered cases of sickness, with nervous symptoms appearing in series among farm horses during the hottest months of the year, are most likely due to an infectious disease and probably not to botulism. The action of spoiled or mouldy food or poisonous plants can usually be excluded on epizoological grounds." On autopsy, characteristic microscopic lesions in the central nervous system are found in encephalitis, while it is highly improbable that either gross or microscopic lesions are found in the central nervous system in botulism. Hemiplegia is one form of paralysis that is suggestive of a brain lesion; this has been observed in degenerative changes affecting one hemisphere, in brain tumor affecting one hemisphere, and in cholesteatoma. A right-sided paralysis indicates a lesion in the left hemisphere of the cerebrum, the paralysis being on the side opposite that of the lesion.

A laboratory diagnosis of infectious encephalitis may be made by means of animal inoculation; for this purpose it is necessary to have a fresh brain. Hurt[16] has reported a case in a colt which presented symptoms and microscopic brain lesions characteristic of virus encephalitis, yet proved on animal inoculation to be affected with rabies.

Prophylaxis.—Embryonic-chick-tissue vaccine, two injections at intervals of seven to ten days, is believed to confer a solid immunity for at least six months. In a release by the U. S. Bureau of Animal Industry, it is reported that among animals vaccinated with embryonic-chick-tissue vaccine in Minnesota in 1938 there was an incidence of 4.5 per thousand in contrast with 36.6 per thousand among nonvaccinated horses and mules. In Iowa the incidence was 5 in contrast with 72. The superiority of chick-tissue vaccine over brain-tissue vaccine

has been definitely established. The production of this vaccine is based on the report of Woodruff and Goodpasture,[17] that the chorio-allantois of developing chicken eggs is an ideal substance on which to grow virus.

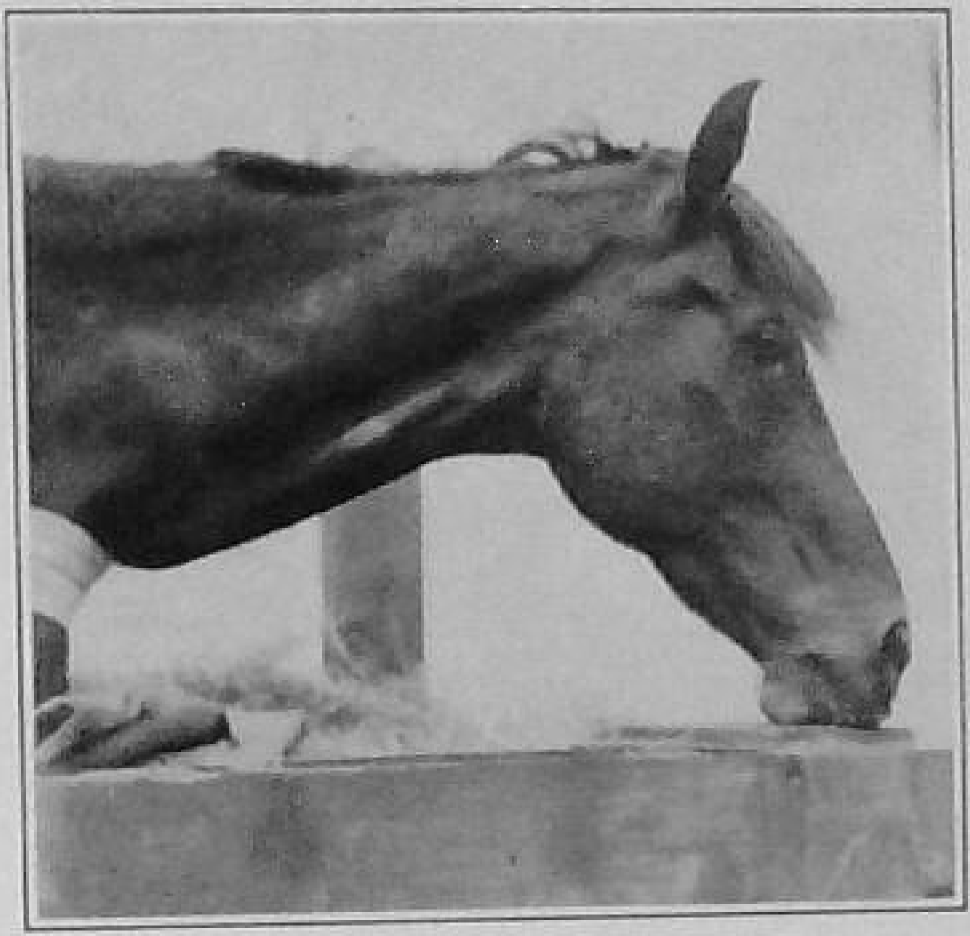

Fig. 82.—A rest for the head to keep it level with the body is recommended.

In the use of the *intradermal* method of vaccination, undesirable reactions have been avoided without any decrease in the degree of immunity (Schoening et al.[18] 1940).

Reactions have been encountered in some horses injected during the period of an epidemic, and acute clinical cases have developed a few days after the injection. Such reactions are thought to be due to superimposing a virus injection on a subclinical natural infection, or on a severe natural infection which was not recognized at the time. Since at least fifteen days are required to produce immunity, the vaccine is most effective when given before an outbreak of the disease, and this should be not less than four to six weeks prior to the date when the first natural cases may be expected.

Concerning the need for vaccination of horses, the following advice has been supplied by Schoening:[18] "There is no need for vaccinating horses that are in areas where the disease has not appeared until and unless it does appear there, for experience last year showed that if animals were vaccinated twice, at several day intervals, as soon as the disease appeared within 15 or 20 miles, they were protected, and losses were virtually nil." Efforts to prevent infection by protecting farm

Note.—The cuts and legends on encephalomyelitis are printed through the courtesy of the North American Veterinarian from an article on "An Infectious Brain Disease of Horses (Encephalomyelitis)" by Haring, Howarth and Meyer, October 1931, and Cir. 322, Univ. Calif., Berkeley.

horses from the bites of insects are of doubtful value. Antiserum provides almost immediate protection, but it lasts only two to three weeks, and is said to leave the animal with an increased susceptibility.

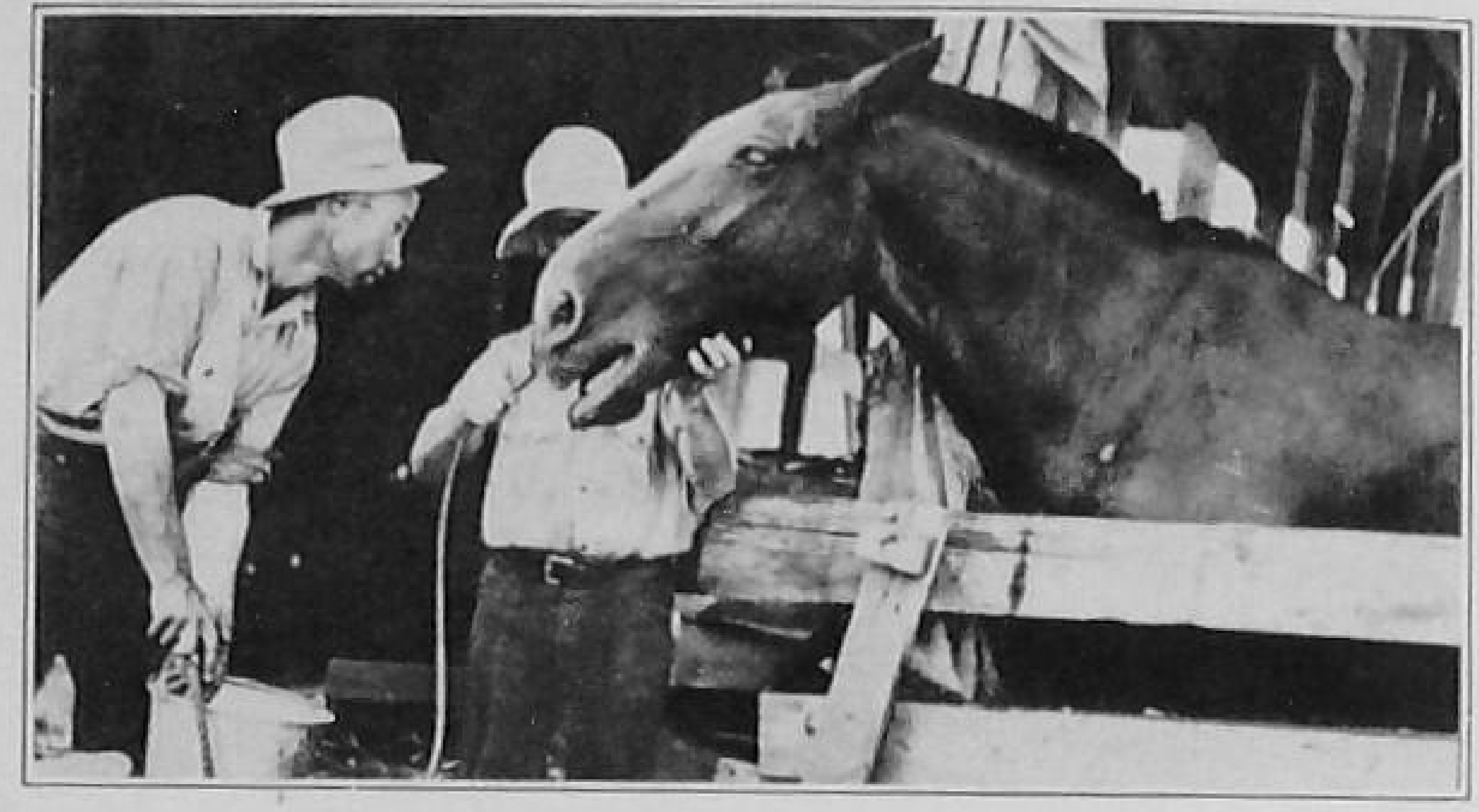

Fig. 83.—Assisting a thirsty horse to drink. Some of the horses which are unable to drink from a pail swallow eagerly when water is injected slowly into the mouth.

Treatment.—Treatment of the sick is entirely symptomatic. If the animal is unsteady on its limbs, but still able to stand, the use of slings or similar support is recommended. Anti-equine encephalomyelitis serum (250 cc.) is of doubtful value. Campbell[19] states that he used large amounts in 1937 and none whatever in 1938 without any change in the mortality. Provide a constant supply of clean water and any food that the animal will eat. When swallowing is difficult water may be given twice daily through a stomach tube, and this may be supplemented with physiological saline solution (1000 cc. daily per vein) or dextrose 40 per cent solution (250-500 cc. daily per vein). No effective remedy for equine encephalomyelitis has been found. In outbreaks where the mortality is 50 per cent or less, individual cases undoubtedly derive benefit from attention to comfort, nourishment, and general care.

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RABIES

(Hydrophobia; Lyssa)

Definition.—An acute fatal encephalitis characterized by deranged consciousness and paralysis. The virus is filterable and is transmitted chiefly by the bite of a dog.

In recent years infection has spread to wild animals and, bites from foxes, skunks, and coyotes have become a frequent cause of rabies in domestic animals.

Etiology.—Rabies is world-wide in distribution in dogs, cats, and carnivora in general—wolves, foxes, coyotes, and it is transmitted to herbivorous animals, and man by the bites of these animals. The two principal vectors are the dog and fox. Since they penetrate more deeply, the bites of wild animals are more dangerous than those of dogs.

The following countries are reported to be free from rabies: Great Britain, Ireland, Denmark, Norway, Sweden, Holland, Australia, and Hawaii. The frequency of the disease is in direct proportion to the efficiency of the sanitary regulations of the community. It is common in Russia, adjacent European states, and the United States.

In the United States the number of cases of rabies in each species each year, compiled from a questionnaire sent to the livestock official and the health officer in each state, is published in the Annual Report of the U. S. Bureau of Animal Industry. And additional information is published in the Annual Proceedings of the U. S. Livestock Sanitary Association. In the four-year period from 1947-50 the following statistics have been compiled:

	Dogs	Man	Cattle	Sheep	Swine	Cats	Miscellaneous, mostly wild animals	TOTAL
1947	6,949	26	766	15	20	393	728	7,597
1950	4,979	9	948	48	85	428	1375	7,910

The gradual decrease in the number of rabid dogs each year, and a decline in the occurrence of rabies in man, is attributed to the Nation-wide program to control rabies, and the community-wide vaccination of dogs. Yet the Chairman of the Committee[1] on Rabies reported before the Livestock Sanitary Association in 1951 that "The rabies situation is at a standstill or getting worse, despite the fact that there were a few less cases this year than in previous years. New territory has been invaded, and the incidence of the disease in a number of states, where it has appeared over the years is still high." With the exception of New England, rabies is reported chiefly from states within an area bordering on the Atlantic, the Gulf of Mexico and the Mississippi River, with Texas, New York and Kentucky leading in numbers. West of this area rabies is most prevalent in Oklahoma, Colorado, and California, with little or none reported from the Northwest.

The virus is most readily found in the brain and medulla of animals dead of the disease, and it may be present in the lachrymal glands,

udder, testicles, and kidneys. In the living animal it is present in the salivary glands and saliva from one or two days before symptoms appear until death. The presence of Negri bodies in the hippocampus is pathognomonic of the disease. Cultivation and isolation of the virus has not been accomplished, and the nature of the Negri bodies has not been determined. The virus is highly resistant when dried quickly in nerve tissue; it succumbs quickly to putrefaction, and retains its virulence for a long time in glycerine.

Pathology.—The only characteristic lesions are microscopic. The most important from the standpoint of early diagnosis are the *Negri bodies* in the cytoplasm of the large nerve cells; these are sharply defined round or oval structures that are most abundant in the cells of the hippocampus major; they are also present in the cells of the cortex of the cerebrum and cerebellum and the spinal ganglia. Other lesions in animals that have died of rabies, but are absent in the early stages, are changes in the peripheral cerebrospinal ganglia, described by Gehuchten and Nelis. These consist in the destruction of the normal large nerve cells, which are replaced by small round cells.

In animals that have died of rabbies the *Gasserian ganglia* may disclose lesions that are characteristic, but not pathognomonic, of the disease. An inflammation of the capsule of the ganglia cells leads to the replacement of these cells by those arising from the inflammatory process—leucocytes, epitheliod, lymphoid, and mast cells. Similar changes occur in dog distemper, while they may be absent in the early stages of rabies. Frothingham[2] found these lesions of diagnostic value when the brain had been destroyed by injury or putrefaction.

Symptoms.—The incubation period is relatively short when the wound is located near the central nervous system, as on the face and head, when it is a deep puncture, when the tissues are rich in nerves and lymphatics, when the virus is rich in virulence and quantity, and in young animals or children. Bites on the lips and nose are especially dangerous. The disease is said to be most common in the late winter and spring because stray dogs range the country in search of food and mates.

Only a limited number of those bitten by rabid dogs become affected by the disease; this is estimated in man at 15 per cent and in animals at from 20 to 30 per cent.

Stafford and Phillips[3] report that dogs exposed to rabies escaped, whereas there would be as much as 25 per cent mortality in cattle and other livestock due to rabies after exposure, even after treatment.

The average period of incubation for man and different species of animals is: dog, 3-6 weeks; horse and cow, 2-10 weeks; sheep and goat,

3-4 weeks; swine, 2-3 weeks; man, 3-9 weeks. In rare instances the period of incubation may be as long as three months; it varies widely in all species.

There are two distinct signs of rabies: deranged consciousness, and paralysis. The symptoms vary somewhat in different species and individuals. Occasionally the initial period of deranged consciousness is missing; the animal merely shows paralysis, *dumb rabies*, in contrast to the more *furious* form. Three distinct stages mark the typical course:

(a) Premonitory Stage, in which the dog is depressed, restless, irritable, and avoids association with people. Increased reflex irritability may be shown by increased friendliness to acquaintances and a tendency to bite strangers. There may be a perverted appetite, manifested by refusal of food, and licking or eating all manner of indigestible substances, such as straw, dirt, wood, stones, glass, etc. Intense itching, with licking or biting of the part, may develop at the seat of the bite; this is most often observed in horses. At times the appearance of the animal may be apparently normal.

(b) Stage of Excitement.—This is characterized by excitement, restlessness, and vicious or aimless attacks on any moving object or animal. The dog may disappear and wander aimlessly over the country, biting other dogs, cattle, or any animal that crosses his path. Infrequently the special object of the rabid dog's attack may be an animal with which it has been closely associated. In cattle this period is marked by bellowing, tenesmus, and clonic spasms, such as jerking movements of the legs. Dogs may show hallucination by snapping at imaginary flies. There is also a marked change in the sound of the bark of a rabid dog; it has been described as a hoarse howl. This stage lasts for three or four days, and towards the end paralytic symptoms appear.

(c) Paralytic Stage.—In the dog the usual early paralytic signs are drooling and an open mouth, caused by paralysis of the lower jaw; in localities where rabies has made its first appearance this symptom has given it the name of "drop-jaw disease." Other early paralytic signs are ptosis, strabismus, a staring expression, and inability to swallow. Efforts to swallow cause spasms of the throat muscles, a reasonable explanation for the "hydrophobia." After paralysis once appears it develops rather rapidly, soon involving the body, hind parts, tail, bladder and rectum, and terminating fatally in exhaustion on the fifth to the eighth day after the onset of the first symptoms. In the paralytic form, without previous excitement, the attack is fatal on the second or third day. In the later stages, there may be a medium grade fever and a rapid pulse.

Cats present symptoms much like those in the dog, except there is less tendency to wander.

In the *horse,* often the first symptom is in intense itching of the bitten place (lips, nose), which causes extreme rubbing. Fear and restlessness are obvious. The animals stare, paw, and grab at the manger with the teeth, and move the ears continually. With the development of mania the horse attacks other animals and man and may direct the attack against some particular individual. The periods between such attacks are variable. They may bite themselves, tearing out parts of the flesh, or bite viciously on parts of the stall, injuring the mouth and breaking the teeth. As in other species, diagnosis in the early stages may be difficult because of absence of excitation, especially at the onset, between paroxysmal attacks, and even during the entire course (paralytic type). Finally, paralysis is general, the end coming on the fifth to eighth day, as in the dog. Rabies in a horse has been diagnosed clinically as encephalomyelitis; as described by Hurt[4] there were facial paralysis, tendency to "wander," pressing the head against solid objects, inability to swallow, and a rapidly developing general paralysis. The violent form of infectious hepatitis in the horse has been diagnosed as rabies; it is differentiated by the intense yellow color of the mucous membranes (infective jaundice).

Cattle are restless, excitable, and aggressive, though at first these symptoms may be intermittent and somewhat indefinite. They stand in one place, raise and lower the head, retract the upper lip, bore with the horns, and show clonic spasms of the muscles of the limbs by sudden jerking movements of the legs. There may be periodic attacks of excitement when they exert every effort to break loose from the stanchion; between such spells they are apparently normal. In most cases there is a prolonged hoarse bellow. They rub and bite, salivate, and grate the teeth. The digestive symptoms are anorexia, suspended rumination, tympany, inability to swallow, and impaction of the rumen with extreme straining. The tympany and other symptoms of choke have led to suspicion of a foreign body in the throat with a consequent manual exploration, laceration of the hand, and a badly frightened subject for Pasteur treatment. Other symptoms are clonic spasms of the neck muscles, sexual excitement, and switching the tail. The initial paralysis is in the throat or hind parts, leading to death in the usual course of 12 to 13 days. In the bull, prolapse of the penis has been observed. Violent attacks may be aroused by the presence of a dog.

Cushing[5] has described a series of cases of rabies in cows in which the diagnosis was difficult because of the absence of excitation, motor

irritation or even paralyis until near the end. He writes of two heifers whose brains showed positive Negri bodies that "their temperatures were normal, they showed no brain symptoms whatever, had good use of their legs and had normal rumen and intestinal sounds; the outstanding symptom was complete anorexia. These animals lived 3 or 4 days and died without showing any straining or viciousness, although toward the end they were unable to rise."

Fox and Roberts[15] have described some of the difficulties in making a clinical diagnosis of rabies in cattle.

In *sheep* the symptoms are similar to those in cattle, though excitement is often wanting. Excitement is manifested by restlessness, stamping the feet, and marked sexual desire shown by riding other animals. Gnawing and licking of the wound are common.

Swine attack other animals, even their own young when excited. They hide in the straw, gnaw at the wound, and soon become paralyzed.

Course and Prognosis.—The usual course in the dog is from four to seven days. Ten days is set as the limit beyond which a dog sick of rabies cannot live. Recoveries have been reported, even of dogs whose victims die of rabies, but such terminations are extremely rare. Mules die in from 18 to 36 hours after the onset.[3]

Diagnosis.—The *clinical* diagnosis is not difficult if the course has been typical and the observation complete. In dogs the changed attitude, aimless wanderings, unprovoked attacks, and rapidly fatal course, are almost pathognomonic. The paralytic or dumb form may be confused with other forms of encephalitis. The dropped lower jaw and changed voice are especially significant. Added to these are the negative autopsy findings with the presence of unusual foreign material in the stomach. Suspected animals should not be destroyed; if at the end of two weeks a suspected dog is alive and well, the possibility of rabies may be dismissed.

The presence of *Negri bodies* in smears of brain tissue is positive evidence of rabies. For laboratory examination for Negri bodies, ship the entire head well packed in ice, or the brain preserved in glycerin. A negative result is not entirely conclusive; if the animal has been killed in the early stages of the disease they may be absent, though usually they are present. This is the most rapid method of diagnosis of rabies. A mouse inoculation test devised in the laboratories of the Rockefeller Institute by Webster[6] is a more reliable detector of rabies virus than the Negri body test. In Alabama, in 1937, the percentage of specimens microscopically negative and found positive on mouse injection was 12.6, while the percentage microscopically positive and found negative on mouse injection was 4.0 (Damon and Stellars).[7] Character-

istic symptoms develop in from six to ten days after exposure, and vaccination should begin within a week following the bite.

Control.—Little can be added to the statement by Moore[8] in 1916, that "the eradication of rabies infection resolves itself into two procedures. (*a*) The destruction of all ownerless and vagrant dogs, and (*b*) the muzzling of all dogs that appear upon the streets or in public places. In thus preventing the propagation of the virus, as shown by the results obtained in Germany and Great Britain, the disease will be practically exterminated." Current recommendation for control of rabies in the United States provide a dog quarantine in a township, county, or city for not less than thirty days or until released by the authorities. This is supplemented by free vaccination of dogs which may be released from quarantine after a "designated" period, a quarantine of three to six months on unvaccinated dogs, while stray and ownerless dogs are impounded or destroyed. Since dogs under 6 months of age are not readily immunized, they should be confined until the quarantine is released. It is generally conceded that improved vaccines now available for dogs confer an immunity for one year after a single injection. As reported by the Committee on Rabies,[9] "the alarming increase in certain areas has forced the employment of more active control measures," but with the passing of the alarm, the force is released and the control measures are soon forgotten. Rabies cannot be eradicated from the United States until a uniform program under the supervision of a single agency is obtained (Johnson).[10] A recent additional problem is the increase in rabies in wild animals.

Prophylaxis.—Pasteur was the first successfully to vaccinate people who had been bitten by rabid dogs. The *Pasteur vaccine* consists of attenuated virus prepared by drying the spinal cord. The virus from a naturally infected dog is termed *street virus*. If such virus is passed through several rabbits it acquires an increased and a fixed virulence (*virus fixe*). When a rabbit receives a subdural inoculation of the brain of a rabid dog, the period of incubation is from fifteen to twenty days; when fixed virus is used, the period is reduced to seven days. The virus in spinal cords of rabbits inoculated with fixed virus gradually loses its virulence when preserved in dry air, and by such exposure various definite strengths of virus are obtained. Immunity is established by at first injecting a highly attenuated virus, and on successive days using a slightly more virulent form.

Avianized vaccine is a chick embryo-adapted strain described in 1953 by Cox[11] and distributed with the claim that it is highly stable, eliminates occurrence of postvaccinal paralysis and induces a high degree of immunity. The recent decrease in rabies in dogs and man is attributed

to the extensive prophylactic vaccination of dogs.

Vaccination of cows that may have been exposed to fox and dog bites is expensive, and the dosage has not yet been established. In a report by Rollins,[12] eight bitten cattle weighing not over 800 pounds received 20 to 35 cc. repeated three times at two-day intervals and none contracted rabies. He reports that most animals bitten in the nose develop rabies in about 21 days and losses in vaccinates have run as high as 25 per cent. Four cows, bitten in the nose, remained normal when injected with 100 cc. followed by two subsequent doses of 50 cc. at two-day intervals: this dosage is commonly advised.

In human practice treatment with hyperimmune antirabies serum combined with a course of vaccine has been recommended for field trials where intensive vaccine treatment protects less than 70 per cent, as in Iran following bites of wolves.[13]

That vaccination against rabies is not always successful is shown by the following statement by Dr. Frank Jirka,[14] Director of Public Health of Illinois: "In spite of the institution of prompt and adequate vaccination against rabies, between 0.5 and 1 per cent of persons bitten by rabid animals contract rabies. This means that while vaccination against rabies in humans is an exceedingly valuable method of preventing rabies, it is not 100 per cent effective. The only method which will assure the eradication of rabies is the disposal of stray dogs and the confinement of home-owned dogs until rabies has disappeared from the community."

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PSEUDORABIES

(Mad Itch; Infectious Bulbar Paralysis; Aujezsky's Disease)

Definition.—Pseudorabies in cattle is an infrequent noncontagious fatal enzootic caused by a filterable virus, and characterized by intense pruritus, paralysis, and death in from twelve to eighteen hours. It occurs in swine as a relatively mild, highly contagious disease. It has been described in Hungary, Brazil and Siberia in dogs, cats, cattle, swine, and rats. One outbreak in this country occurring in August 1930 in Iowa has been described by Shope,[1] who states that the disease is so rare as to constitute a veterinary curiosity. According to Murray,[2] of the Iowa State College, the condition known for many years as "mad itch" has been regarded by many as the skin form of hemorrhagic septicemia; this statement indicates that the disease is not of recent origin in the Middle West, and that limited enzootics are occasionally met with. In a description of an outbreak of the disease on a farm in Iowa by Rossing,[3] there are reports from several veterinarians who had

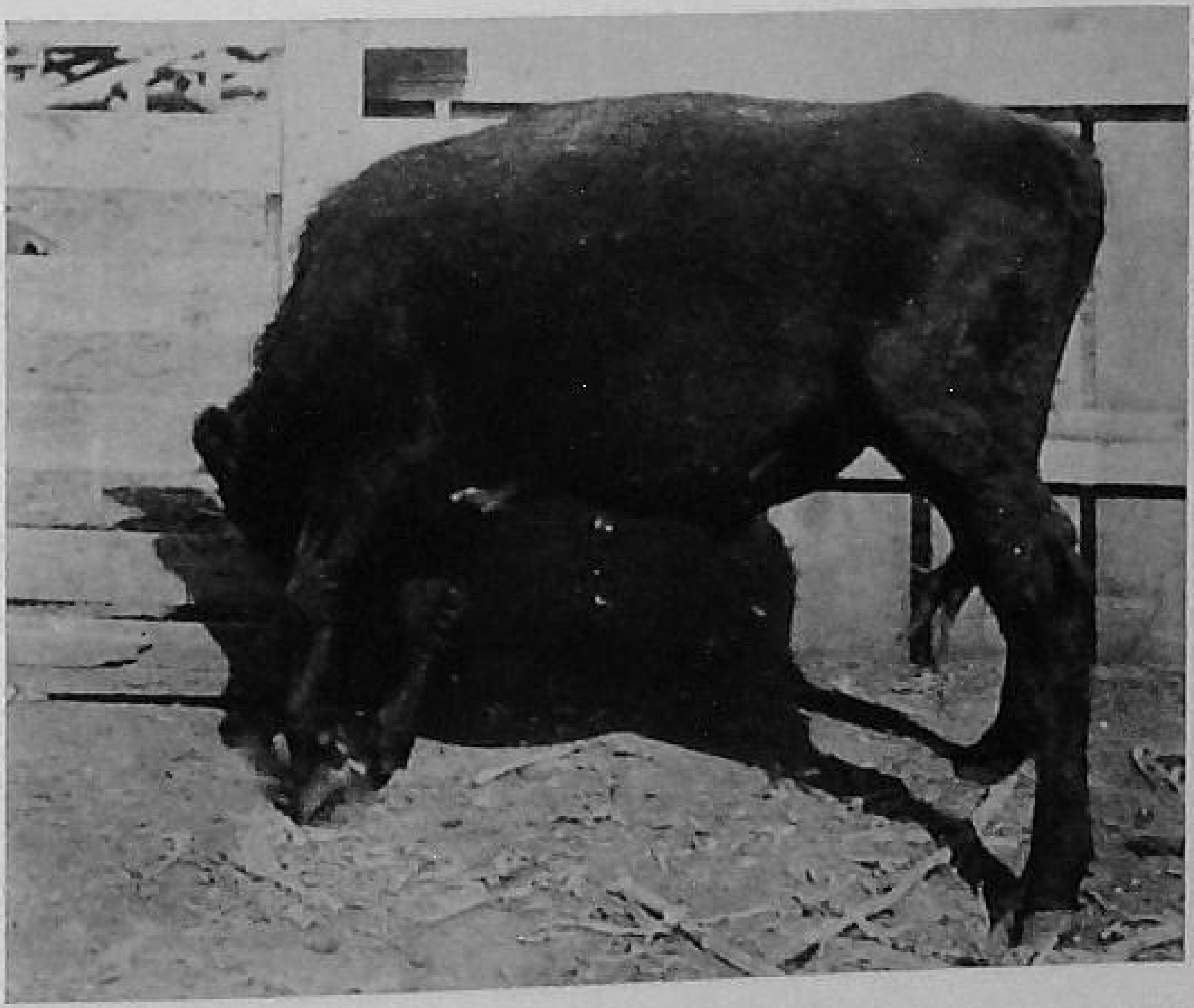

Fig. 84.—Pseudorabies (Mad Itch). (Photograph by courtesy of Dr. Charles Murray, Ames, Iowa.)

seen from one to a dozen or more outbreaks in cattle in Iowa, and always in animals that had mingled with swine.

That swine may be important in the spread of the disease is suggested by the report of Köves and Hirt[4] that unrecognized widespread distribution in swine has existed for at least twenty years. The morbidity may reach 40 to 60 per cent and the mortality may be as high as 5 per cent. According to their account the virus is widely distributed in the body, the incubation period is from three to five days, and the symptoms are anorexia, vomiting, diarrhea, muscular spasms, involuntary movements, and paralysis of the pharynx with drooling of saliva. Pulmonary edema is found on autopsy, and histological changes are found in the brain tissue.

Etiology.—Pseudorabies was first described by Aujeszky[5] in Hungary in 1902, who reproduced the disease by inoculation of rabbits with material from the medulla of a cow and a dog. His observations were confirmed by others, who demonstrated the susceptibility of rabbits, guinea pigs, rats, mice, carnivora, cattle, sheep, and goats. Horses were found to be highly resistant. The virus was found in greatest abundance at the seat of inoculation; it was also found in the blood and central nervous system.

In the investigation in Iowa by Shope[1] in August 1930, he obtained a filterable virus from the brain of 1 of 9 animals to die in a herd of 12.

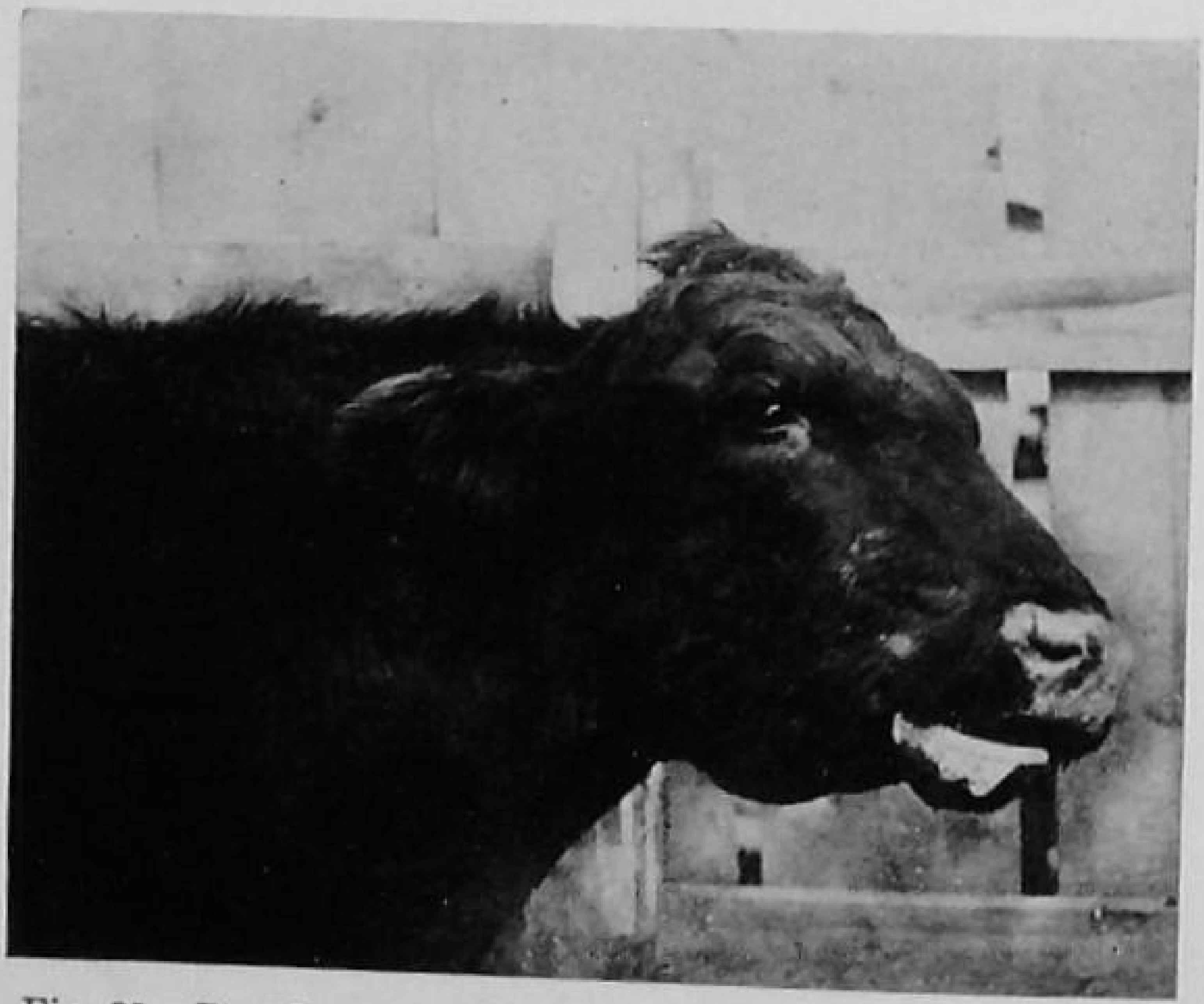

Fig. 85.—Pseudorabies (Mad Itch). (Photograph by courtesy of Dr. Charles Murray, Ames, Iowa.)

Physiological salt solution suspensions of the effective brain when injected subcutaneously into rabbits produced the clinical picture of "mad itch" as seen in cattle. Brains of intracranially infected rabbits were also infective. The virus passed through Chamberland L3 and Berkefeld V, N, and W filters. In rabbits the incubation period varied from one to three days, and the virus was recovered from the site of the inoculation and from the lungs, as well as from the brain; it was not found in the heart, blood, liver, or spleen. This virus was still active after remaining 154 days in 50 per cent glycerol in a refrigerator. A calf inoculated subcutaneously with rabbit brain virus developed typical symptoms of "mad itch" after an incubation period of four days. Other animals successfully inoculated by Shope[1] were cats, mice, ducks, and swine. Under laboratory conditions the disease was not found to be contagious. On the farm where the Iowa outbreak occurred there had been a highly fatal epizootic among rats the preceding week. European writers report that outbreaks in dogs, cats, and cattle are occasionally associated with fatal epizootics of the disease in rats, and they also state that the frequency of pruritus on the face is suggestive of transmission by rat bites. Shope[6] has reported that it is a highly prevalent mild contagious disease in middle western swine, that in these animals the nose serves both for the entrance and the exit of the virus, and that fatal pseudorabies infection in rabbits can be induced merely by bringing their abraded skin into contact with the noses of infected swine. It is believed that the swine may transmit the disease to cattle by transfer of virus on their snouts to the abraded skin. An attack confers immunity.

Morbid Anatomy.—In cattle, persistent rubbing leaves the skin over the thighs and buttocks denuded of hair, dark, leather-like, and smeared with bloody serum. The subcutaneous tissue over the affected parts is thickened with a serous and gelatinous infiltration, but the underlying muscle is not involved. If the animal is recumbent for some time there may be an edema of the lungs. There are no gross lesions of the abdominal and thoracic viscera and of the central nervous system.

Symptoms.—With rare exceptions intense puritus is the first symptom to be observed and this may be combined with a twitching of the muscles of the face and neck. There is a continuous licking of an area on the hind quarters, and after two to three hours the skin is denuded of hair. As the pruritus increases the animals rub violently against posts, barbed wire, or any other firm object; they may bite and gnaw themselves. At the end of twenty-four hours the patient is usually down and unable to rise because of paralysis. Salivation and marked grinding of the teeth are sometimes present. Death occurs within two or three days after the first symptoms appear; it is immediately preceded by convul-

sions, bellowing, and rapid breathing. There is rarely any fever until shortly before death. In the cases described by Rossing[3] the first symptoms were twitching of the tail and weaving movements of the hind parts. After one to three hours there were salivation, and rubbing of the head and upper parts of the neck. The pruritus was so severe that some of the cattle became delirious and attacked attendants. The affected areas were rubbed until they were raw and bleeding. After about six to twelve hours the weaving of the hind parts was pronounced and the animals fell in a spasm and remaind paralyzed until death, which occurred after a course of from eighteen to twenty-four hours. In most cases the pruritus was on the head and neck. Rabbits that have been inoculated subcutaneously develop a fever, scratch at the seat of inoculation, and die in from six to twenty-four hours after the onset of the symptoms. Intracerebral inoculation in rabbits causes death in from twenty-four to fifty hours; they show excitement, run blindly into objects, and develop extreme orthotonus. In three Shorthorn cattle described by Sellers *et al.*,[7] a mild pruritus was observed in two, and the third developed marked bloat and died within 24 hours after the onset without showing pruritus. Diagnosis was based on the results of a subcutaneous injection of rabbits with brain emulsion: death occurred in five days, abrasion and laceration were present at the point of inoculation, and the histologic findings of the brain conformed to those described by Hurst.[8]

Shope concluded that mad itch and pseudorabies are the same, and that the disease observed by him in the United States is identical with that described as pseudorabies by Aujeszky and others, even though the virus in the bodies of experimental animals was found to be more widely distributed than in individuals examined in this country. No remedy has been found. When the disease appears cattle should be separated from swine.

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MALIGNANT HEAD CATARRH

(Malignant Catarrhal Fever)

Definition.—An acute highly fatal infectious disease of cattle characterized by inflammatory edema of the tissues lining the facial sinuses, the nose, and the throat; often the eyes are involved. Nervous symptoms are frequent. It has been described in Europe and Africa, and has frequently been observed in the United States. The disease in Africa, termed "snotsiekte in cattle," is regarded as identical with European form of malignant head catarrh, and possibly it is identical with the form observed in the United States.

Etiology.—Malignant head catarrh is usually a limited enzootic in stabled cows in the spring months. In the vicinity of Ithaca, where it is occasionally sporadic in pastured cows, it has been seen in every month from March to September. In certain districts the disease is endemic and tends to appear each spring; this has been observed in the region of Lake Ontario. In 1926 an extensive and severe epidemic was reported from St. Lawrence County, New York. Marshall[1] and associates have written from Pennsylvania that "it is common in this section of the country." From 1913 to 1927 it was diagnosed in the ambulatory clinic of the New York State Veterinary College on six different occasions, and sporadic cases are met with almost yearly. It is reported that unsanitary conditions, low altitude, and moisture favor the development of the disease, but there is little evidence to support such a view or to indicate that predisposing causes exist. Often the sick are in widely separated parts of the stable, a condition that suggests absence of spread of infection, either by direct or indirect contact. Heavy losses from malignant head catarrh have been reported from North Dakota.[2]

Mettam[3] studied the disease extensively in East Africa, and was able to show that cattle contracted it from association with apparently normal herds of wildebeest, that when wildebeest calves suckled on a cow the foster-mother contracted the disease, and all cattle in contact contracted the disease and died. Under natural conditions the period of incubation appeared to be about one month, and the course was from four to ten days, ending in death. He reached the following conclusions:

"1. The etiological agent is an ultra-visible but non-filterable virus closely associated with the erythrocytes.

"2. The disease is transmissible in series through cattle by the subinoculation of large quantities of blood (5-200 cc.).

"3. The disease is not transmissible from sick to healthy cattle by contact, nor did infection follow the ingestion of virulent material.

"4. Although the wildebeest may harbor the virus they have never been observed to show any clinical symptoms of infection. All wildebeest are not reservoirs of the virus and the percentage of carriers in a herd is not known.

"5. Under natural conditions the disease is conveyed from wildebeest to cattle by some blood-sucking insect." The fencing of farms, the limitation of movement of wild game, and the decrease in the number of herds of wildebeest have been followed by the disappearance of snotsiekte in South Africa—Du Toit and Alexander.[4]

Götze and Liess[5] have reported that in Germany malignant head catarrh breaks out in cattle when cattle and sheep are associated together in the presence of an unknown vector, though sheep do not have the disease. Götze transmitted the disease by blood transfusion from sick to heathy cattle in 13 out of 34 cases. The period of incubation was from sixteen days to ten months; he also reported carrying the disease through four serial passages in cattle. The view expressed by Götze that malignant head catarrh is acquired by cattle through contact with sheep, is not generally accepted. This has been expressed by Du Toit in the statement that "a statistical survey of the available evidence indicates that sheep cannot be considered to play any significant rôle." Extensive statistics upon this phase of the etiology have been collected by Wyssmann,[6] who also concluded that sheep are not carriers of the infection.

Daubney and Hudson[7] reported in 1936 on transmission experiments in Kenya with a "mild" strain and a "head-and-eye" strain of infection. The mild strain was irregularly transmissible by blood inoculation after a relatively long incubation period. The virulent head-and-eye strain was transmitted by blood, brain, and gland inoculation, "the disease being reproduced with uniform regularity and an exceedingly high mortality." The period of incubation in 23 of 33 cattle was from sixteen to twenty-four days; the maximum was sixty days in one case. Only one case of contact infection was observed. The disease was transmitted from cattle to rabbits by subdural inoculation of brain tissue and by intraperitoneal inoculation of blood. The disease was also "reproduced in cattle in typical fatal form by the inoculation of material derived from rabbit passages." After numerous failures to transmit malignant head catarrh in Kenya, S. Africa, Piercy[8] finally was successful, using a mixture of spleen, brain, kidney and prescapular gland

which was injected into the prescapular gland of a bovine. The incubation period varied from 14 to 37 days. Cattle were also infected by the intravenous, intradermal, intramuscular, and subcutaneous routes. One animal was infected with 0.05 cc. of concentrated white cell inoculum, which suggests that the circulating virus is attached to the white cells. In an effort to stem the spread of the disease, a formalinized vaccine was used with apparently successful results. There seems every reason to suppose that the virus is attached to cells of the lymphocytic series and there are good grounds for believing that transmission is effected by a blood-sucking insect.

From the available information one is justified in the conclusions that the disease is caused by a virus, some strains of which are highly infective; that it is transmissible experimentally by the injection of relatively large quantities of infective blood; and that the mode of natural infection is unknown.

Morbid Anatomy.—The mucosa of the pharynx and nose is intensely inflamed; it is almost black, thickly edematous, and hemorrhagic. A purulent and gelatinous exudate often covers the surface. Necrosis and ulcers are common. These changes often extend to the mucosa of the nasal septum, the turbinated bones, ethmoid cells, and facial sinuses. The lymph glands around the head are swollen and hemorrhagic. The meninges of the brain are highly congested. The eye may show turbidity of the cornea and the anterior chamber may contain a fibrinous exudate. The body cavities sometimes contain a little reddish serum. The kidneys and liver are degenerated and they may be hemorrhagic. Petechiae in the omentum, the mesentry, and the serosa of the heart have been observed. It is not unusual to find swellings and congestion of the lymph glands throughout the body. Various degrees of inflammation of the digestive and respiratory tracts have been found. There are wide variations in the degree and extent of the autopsy findings; any of the changes that characterize septicemia or toxemia may be present.

Symptoms.—The period of incubation is estimated at from two to four weeks. In a typical form the onset is sudden. A cow apparently well at night may be severely sick in the morning. The initial symptoms are marked prostration, dry muzzle, slight extension of the head from soreness of the throat, staring expression, lack of appetite, and almost complete cessation of the milk flow. The skin is dry, the hair rough, and a papillary eruption marked by small elevated tufts of hair may extend over the region of the loins. Local or general trembling is often present and it is aggravated by examination of the patient. The visible mucous membranes are congested and lachrymation and swelling of the

lids are often present. The pulse is 70 to 100, the respirations 25 to 30, and the temperature 105° to 107° F. Stenosis of the nasal passages often causes an inspiratory snoring sound. At the nose one finds a bilateral mucopurulent discharge that is either reddish or yellowish in color and in one or two days becomes fetid. The severe inflammation may extend to the muzzle and lips. Eating and drinking are slow and swallowing movements are frequently observed. In the submaxillary region an edematous swelling is often present; the throat is painful to swallowing and to the touch, and a suppressed painful cough may be induced. In one case the eyes may remain normal; in another the lids may swell badly; while in a third there may be closing of the pupils, bulging forward of the iris, nystagmus, and total loss of sight. When the cow is disturbed, muscular twitching often appears. Other nervous symptoms are excitement, convulsions, hyperesthesia of the skin of the head and neck, monoplegia involving an ear or an eyelid, uncertain gait, paresis, standing with the head pressed against the wall, and other forms of motor irritation. In one of our cases the cow attacked the owner, causing severe injury. The feces show signs of gastrointestinal irritation in the form of a fetid diarrhea, or there may be paresis of the digestive tract with constipation. In the cases described by Marshall severe respiratory symptoms were present. There is rapid loss in condition. The pulse and respiration increase, while the temperature falls. Improvement is apparent at times, but finally the animal goes down and death usually follows within twenty-four hours. The course is from three to seven days and recoveries are rare.

Towards the end of an outbreak, some of the cases may show mild symptoms and be apparently well within twenty-four hours. When the lesions are less extensive the course may be prolonged for a month or more in an atypical form. In these, the suggestive symptoms are fever, papillary eruption of the skin with tufting of the hair, dull hair coat, dry skin, affection of the eyes, discharge from both nostrils, marked weakness, and rapid loss of condition. In such cases the visible autopsy lesions are suppuration and necrosis of the mucosa of the nose and adjacent parts.

Diagnosis.—The disease has been mistaken for hemorrhagic septicemia, inflammation of the eyes, dermatitis, encephalitis, and sublingual abscess. Because of lack of study of this disease in the United States, it is not possible to determine its relation to malignant head catarrh in Europe and snotsiekte in South Africa. Götze[5] described four clinical forms in Germany: a peracute type, leading to early death; an intestinal form which is usually fatal within ten days; a head-and-eye type lasting from one to three weeks and sometimes associated with

the intestinal form; and a mild form in which recovery is the rule.

Treatment.—Symptomatic treatment has proved of little value, but a limited use of large doses of penicillin and streptomycin has apparently been beneficial.

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ULCERATIVE STOMATITIS AND ESOPHAGITIS

(Virus Diarrhea)

In March, 1946, there appeared on eight farms in the vicinity of Ithaca an acute, febrile, contagious disease of cattle, characterized by diarrhea, ulcerative stomatitis, a short course and a low mortality. It has been described by Dr. Olafson and associates[1] as "An Apparently New Transmissible Disease of Cattle." They report that "it can be transmitted to susceptible animals by drenching them with fecal material, subcutaneous injection of blood collected from infected animals during the stage of high temperature, or with splenic emulsion from animals destroyed during the high temperature stage," and "unless extreme care is used it can be carried from farm to farm." Following dosage with fecal material the period of incubation was 22 days. After experimental exposure, heifers 8 to 14 months of age developed a fever in 7 to 9 days, and a leucopenia after 4 to 5 days.

Morbid Anatomy.—As described by Olafson, the characteristic lesions are ulcers on any part of the oral mucosa, occasionally on the muzzle and nostrils, and ulcers in the pharynx and larynx, sometimes with diffuse necrosis. The mucosa of the esophagus is covered with irregular, punched-out ulcers of varying sizes and shapes, and these

are accepted as pathognomic of virus diarrhea since they have not been found in any other disease. In the stomach, omasum, small intestine, and cecum there may be redness or hemorrhages and ulcers. The carcass is emaciated, and a calf may die without developing ulcers.

Symptoms.—The usual severe form is ushered in suddenly with depression, complete loss of appetite and milk secretion, a temperature of 104° to 106°F., a fast pulse, 100 to 120 in a few, and a brownish, watery, fetid diarrhea. These general symptoms are followed almost immediately, perhaps overnight, by an ulcerative eruption on the mucous membranes of the mouth, pharynx, and sometimes the nose. The gums of the lower incisors may be either pink or red, and salivation is abundant. On the muzzle there may be an ulcer-like surface covered with necrotic material; this, combined with a mucopurulent nasal discharge, presents the appearance of malignant head catarrh. In the severe attacks there may be sunken eyes, dehydration, and weakness. The visible ulcers may be widely distributed on the tongue, gums, hard palate, cheeks, and pharynx. The most severe attacks are in cows about to freshen and in calves a few weeks old. In any herd the disease may vary widely in severity from those that are slightly below normal in appetite and milk production to individuals showing weakness, emaciation, and all of the distinctive symptoms of the disease. There is also considerable variation in the number affected in different herds, from a few to 90 per cent, as well as in the mouth lesions which may be present in only a few, or nearly all, in a given outbreak. The changes in the blood are held to be characteristic in the form of a decrease in the number of multinuclear white blood cells, a leucopenia. A serious sequella is the occurrence of abortion following recovery, and there are subclinical forms, for among the aborters are cows that have shown no previous symptoms. Fatal septic metritis has been observed when the abortions occur during convalescence or shortly after recovery; the abortions may be distributed over several months.

In spite of the severity of the attack, even the severe forms usually recover promptly after a course of two or three days, and the mouth lesions recede as rapidly as they developed. With the appearance of diarrhea the temperature usually falls, and the outbreak is over in about a week. While the course is short and recovery is the rule, losses from interrupted production and postconvalescent abortions are heavy.

Diagnosis.—In the first herd to be affected, that of a cattle dealer, there were numerous cases of pneumonia, which apparently is not present in an uncomplicated form of this disease. In some individuals, however, the high temperature and fast breathing suggest pneumonia,

and a diagnosis of either the pneumonic or intestinal form of hemorrhagic septicemia is liable to be made, especially in the beginning of the outbreak. It might also be diagnosed as some unknown chemical or plant poisoning, and for a time in the current outbreak, rinderpest was suspected. The diarrhea may at first suggest winter dysentery. The pathognomonic significance of ulcers on the mucosa of the esophagus has been considered, but ulcers on the mucosa of the esophagus have been observed in aphthous stomatitis, and in malignant head catarrh. Individual cases resemble malignant head catarrh, but in malignant head catarrh the mortality is high and usually there is turbidity of the cornea. There is a resemblance between the early stages of hyperkeratosis and virus diarrhea, such as the pink color of the gums of the lower incisors; but hyperkeratosis is chronic, the mortality is high, and there is severe involvement of the skin.

Treatment.—Of the various treatments used, such as penicillin, the sulfonamides, and symptomatic, blood transfusion was the only one to be apparently beneficial.

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COWPOX

(Variola; Vaccinia)

Definition.—An acute contagious eruption of the teats and udder of cows which passes through four stages: papules, vesicles, pustule, and scab. The corresponding disease in man (*Variola*), and in sheep (*Variola ovina*) is chiefly a general eruption. In the horse (*Variola equina*) the eruption is local. De Jong[1] reports that contagious pustular stomatitis of the horse is the most frequent form of Jenner's horsepox. Swine pox (*Variola suilla*) has been described in the United States by McNutt,[2] Murray, and Purwin as a highly infectious, contagious affection of young pigs. It is a benign affection, not characterized by vesicles and pustules, as observed in cowpox and smallpox. It is believed that the various forms of pox (vaccinia, ovina, etc.) have been acquired from a common source (variola) by repeated passage through the different species of animals.

Cowpox is the chief form of this disease in the United States. Historically it is of importance because the practice of inoculation of man with virus of cowpox has nearly eliminated what was one of the most dreaded scourges of the human race previous to the nineteenth cen-

tury. This was the first successful artificial vaccination against disease.

Etiology.—Genuine cowpox (vaccinia) is of occasional occurrence in the United States, sometimes involving several herds in a neighborhood, and often it is sporadic. It varies in intensity from a mild transient form to a severe, persistent and recurrent type that may persist in the herd for one or two years. Caretakers become infected from cows, and after vaccination against smallpox they may transmit the disease to cows.

The virus is filterable. Little is known of its persistence in the affected tissues. Outside the body it seems to be harbored in stables for an indefinite period, unless disinfection is practiced. Hess[3] writes that in most cases in Switzerland it is impossible to account for cowpox by the spread of virus from the milkers to the cows; he believes with Bang that the virus remains virulent in the stable for a long time and spreads by direct or indirect contact to newly added susceptible animals. It is present in the vesicles, pustules and scabs, and it has a special affinity for the epithelial cells of the skin and mucous membranes. In the vaccinated cornea of a rabbit pathogenic cell inclusions (Guarnieri's bodies —*Cytorrhyctes*) may be found. Cows are infected either by exposure to human pox (variola) or to contact with vaccine of bovine origin (vaccinia).

Osler[4] states of variola virus, "The agent is of unusual tenacity and clings to infected localities." The same opinion is expressed by Law[5] in stating that "spring is the time when primipera are first subjected to the danger from the hands of the milker, and when the cow from the non-infected district is brought into an infected stable for the season's milking."

After cows have been infected from a caretaker who has recently been vaccinated it spreads more or less rapidly through the herd. When caretakers are infected from cows, they may suffer severely from the disease. Such instances have been described by Boerner,[6] Reece,[7] Jenner,[8] and Cathie.[9]

Since recent studies of natural outbreaks of cowpox have failed to reveal vaccinia virus, and artificial transmission has also been negative, it is now believed by some that genuine cowpox is rare and occurs only when there has been contact with vaccinated attendants (Hester).[10] This view is expressed by Christen[11] who reported that in only one of fifteen epidemics could vaccinia virus be demonstrated; the others were termed natural or abortive, an abortive form of genuine cowpox. Thus we have natural and genuine cowpox, and differential diagnosis by symptoms alone is difficult. While experimental transmis-

sion of natural cowpox has been negative, and the essential cause is yet to be demonstrated, it is unmistakably contagious within affected herds. The source of the infection is also unknown, but there have been numerous epidemics after flood conditions that exposed the udders to mud and filth. After a normal cow is added to an affected herd an incubation period of nine days has been observed, and this is the period reported by Christen[11] in experimental transmission with vaccinia. In severe epidemics that involve all of the milking cows in the herd, severe secondary mastitis caused by streptococci is common. In natural cowpox neither vaccination with vaccinia nor the disease itself confers immunity.

Symptoms.—The incubation period is from three to six days. The skin lesions—papules, vesicles, pustules, and scab may all be present at one time because of successive eruptions, and this serial development may continue for months. Usually all of the cows in a herd are affected, but the disease may be sporadic. First there appear on the teats reddish painful papules 6 to 10 millimeters in diameter. In one or two days these change into vesicles; they have a metallic lustre, are whitish or yellowish, round or slightly oval, and often present a pit in the center. In Jenner's description emphasis is given to the "livid or bluish tint so conspicuous in the pustules," and absent from other "pustulous sores" on the teats. They have a mesh-like structure that prevents collapse if a single opening is made, and are in the skin rather than on its surface. If undisturbed by milking the pustules reach maturity in eight to ten days, when they collapse and a scab is formed. This eruption is on the teats, rarely on the ventral surface of the udder, and affects only cows that are in lactation. In the mild (*discrete*) type from one to ten vesicles may form on each teat. In *confluent* pox large areas of the teat present a denuded, fissured, painful, moist, or bleeding surface. Extension to the end of the teat may cause pyogenic infection with severe thelitis and even fatal mastitis. Because of reinfection with pox virus, the disease may run an obstinate and prolonged or recurrent course. In rare instances the eruption appears on the body or limbs in association with general symptoms and loss of condition; in this type severe mastitis usually develops. The characteristic symptoms in cowpox are sometimes difficult to recognize because the lesions are not under continuous observation, and as soon as the vesicles form they are ruptured by the hands of the milker. Cowpox infection in milkers usually develops on the hands or forearms. As a rule an attack of cowpox protects against subsequent exposure. But in Jenner's[8] Inquiry, Case IX, he records three distinct attacks in one man in a period of fifteen years,

and mentions second slight attacks in cows. In my records there is a report of a cow with two distinct attacks a year apart.

In the differential diagnosis between natural and genuine cowpox, Christen[11] reports that in natural cowpox there is more rapid development of vesicles into pustules which may be completely formed within ten hours: the depression on the surface of the vesicle and the surrounding areola or zone of congestion are slight or entirely absent. This form is not usually contagious to man. In genuine cowpox the pustule develops more slowly, reaching maturity in about nine days. Further, the vesicles are characterized by a distinct depression on the surface and encircled by a red zone. It spreads readily to attendants.

Treatment.—For the usual mild form, care and cleanliness in milking, and the application of creolin (3 per cent) are sufficient. If the teats are cracked and sensitive at the time of milking, thoroughly soften the tissues with warm creolin solution or zinc oil (equal parts zinc oxide and olive oil). Especially effective is salicylic acid in glycerin (3 per cent) or in Whitfield's ointment: salicylic acid 2 drams (8 Gm.), benzoic acid 1 dram (4 Gm.), lanolin 6 drams (24 Gm.), and petrolatum to make 2 ounces (60 Gm.). Tincture of iodine in glycerin (equal parts) is also useful. Sulfathiazole ointment is an effective agent against both the primary and secondary lesions of cowpox. In one large herd where there were numerous cases of cowpox, zinc oxide ointment proved to be the most effective of several remedies used. Cerbinol liquid (Pitman-Moore) has also proved to be highly effective in the treatment of cowpox.

Prophylaxis consists in early segregation of infected cows, separate caretakers, and disinfection of the stable after the last case has recovered. Vaccination has proved effective only against vaccinia.

The ends of the teats are the frequent seat of various eruptions in the form of fissures, scabs, and necrosis. Apparently these are caused in part by contact with filth, but they may occur in the absence of such exposure, either singly or in several animals.

It may be difficult to differentiate these conditions from cowpox when the stage of vesicular eruption in the latter is passed. The chief significance of teat eruptions, however, is attached to their proximity to the opening of the teat duct, rather than to the character of the lesion. When scabs or fissures or other lesions form on the end of the teat, prompt and vigorous disinfection is indicated to prevent invasion of the teat canal and loss of the quarter from mastitis. For this purpose one may touch the affected part with a pledget moistened with undiluted carbolic acid and then neutralize with alcohol. Tincture of metaphen and scarlet red oil have also been recommended for the treat-

ment of eruptions involving the ends of the teats. Lesions on the teat, especially at the end, may respond favorably to soaking in hydrogen peroxide, or to a small antiseptic pack held in position with tape. Another useful preparation is a mixture of sulfanilamide 20, sulfathiazole 20, and urea 40; this is distributed under various trade names, such as Reazol, etc. Tyrothricin ointment is excellent.

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CONTAGIOUS PUSTULAR STOMATITIS IN HORSES

(Horsepox; Variola equina)

Definition.—Contagious pustular stomatitis of the horse is characterized by the formation of papules, vesicles, and pustules, from 1 to 5 mm. in diameter on a swollen and congested oral mucous membrane. The chief location is on the mucosa of the lips, gums, cheeks, and tongue, especially in the vicinity of the frenum linguae, as well as on the ventral and lateral surfaces of the tongue. Zwick[1] and others consider the cause to be a form of variola.

Etiology.—The disease is comparatively infrequent in Continental Europe and I have found no record of its occurrence in America. It is chiefly observed as a stable enzootic in young horses, and an attack confers immunity. It is of chief importance because of the wide distribution of the variola and variola-like diseases.

The Virus.—There have been two chief views concerning the nature of the virus of the disease. French authors have regarded it as a form of Jenner's pox, while German authorities have considered it to be some other form of virus. For years it has been known to be experimentally transmissible to man, horses, cattle, sheep, and swine, while Friedberger was successful in transmitting it to the comb of a hen. In 1916 de Jong[2] submitted proof that contagious pustular stomatitis of the horse is actually the most frequent form of Jenner's horsepox, and that the virus passes the Chamberland B and F filters; with such filtrate he was able to reproduce the disease in rabbits, cattle, and man. In the experiments reported by Zwick, he secured the virus from a natural case of pustular stomatitis in the horse. An experimental horse contracted the disease in about four days. On the skin of a calf, typical pox papules and pustules were produced in five to six days. In two rabbits the transmission was successful. From the eruptions on the calf, experimental transmission was made to a sheep, a pig, a dog, two rabbits, a man, and a hen. The specific nature of the virus was proved by finding Guarnieri's bodies in the epithelial cells of an infected cornea of a rabbit.

Symptoms.—The development of the papules, vesicles, and pustules is in series; they are especially abundant at the commissures of the lips and in the region of the frenum linguae. The lips, cheeks, and submaxillary lymph glands may be slightly swollen. There are no general symptoms except a possible slight rise in temperature at the onset. The appetite is somewhat diminished because of pain; there is slight salivation and sometimes an odor. The eruption may spread to the mucous membrane of the nose, especially the nostrils, and the skin may be involved. Exceptionally there are conjunctivitis and turbidity of the cornea. Sometimes the eruption appears in the region of the external genitals in the mare, as well as upon the skin on various parts of the body or limbs. Recovery occurs in from ten to fourteen days.

Diagnosis.—The disease is most readily confused with vesicular stomatitis. In the latter there are no nodules and no pustules.

Treatment consists in the use of mild antiseptics, such as potassium chlorate (2-3%), alum, boric acid, etc.

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CONTAGIOUS ECTHYMA OF SHEEP

(Sore Mouth; Contagious Pustular Dermatitis)

Definition.—An acute infectious disease of lambs and kids characterized by a cutaneous eruption of the lips and caused by a virus. The eruption passes through the stages of papule, vesicle, pustule, and crust. Secondary infections with *Actinomyces necrophorus* are not infrequent. The most serious aspect of the disease in western Texas is attributed by Boughton and Hardy[1] to infestation of the inflamed lips with larvae of the screw worm (*Cochliomia macellaria*). Inability of the lambs to suckle and a consequent loss of condition is one of the most serious aspects of the disease. It has been observed in a mild form in man.

Etiology.—The distribution is worldwide. In America it has been chiefly reported from the sheep-raising states west of the Mississippi from the Dakotas to Mexico, but it is prevalent throughout the country and has been met with in New York State. In Texas it appears in the spring and subsides with the approach of cool weather in the fall. In one county in New York it appeared in May as an eruption on the lips of the lambs and the teats of the ewes. It is reported by Newsom[2] that during some years in Colorado nearly every feeder lamb shows symptoms of the disease, and its appearance soon after shipping is taken as a matter of course. Lambs and kids are chiefly affected, but mature animals are not immune unless they have already had the disease. In sheep over one year of age, however, the attack is always mild.

The virus is in the crusts that form on the lips and it can cause typical lesions when inoculated into the skin of any part of the body. Within the body the virus is limited to the skin lesions. Outside the body it is present in the scabs where it survives the winter. Its longevity on the range is unknown, but it is highly resistant; it has been kept active for as long as fifteen months in the laboratory. Infection does not generally spread from one species to another, and it cannot be transmitted to small experimental animals. When infected crusts are placed on scarified skin, lesions appear in from forty-eight to seventy-two hours, and no other method of infection has been successful. Plurality of virus has not been found. The vesicles and pustules develop in a series over a period of about a week.

Natural infection occurs either by direct or indirect contact with virulent scabs that have dropped from affected lambs or kids and it is said to be able to take place through the unbroken skin. Inoculation follows the introduction of extremely small quantities of virus and it is believed that infection may occur through the water. Once a herd is

affected the disease spreads rapidly. One attack confers immunity.

Symptoms.—The incubation period is from four to seven days. The first symptoms are swollen lips covered with wart-like, moist, brownish gray scabs. Later the scabs are firm and cracked and when removed they leave a bleeding surface. It is at this stage that the blow-fly larvae are deposited on the lips of lambs and kids in western Texas. Under usual conditions the scabs gradually dry and loosen and fall off leaving a normal skin. Except in mild forms the period of eruption is about three weeks. Close observation is required to observe the succession of changes from papules through vesicles, and pustules to the formation of scabs. The eruption may spread to the nostrils and around the eyes. In describing the appearance of the eruption Boughton and Hardy[1] write: "It is noted in the development of the lesion that the small vesicles usually are single, there being little tendency for them to coalesce; they may be very numerous and very close together but it is not until the pustules, developing from the vesicles, rupture and discharge their contents, that they coalesce and form a more or less compact scab."

Complications due to infection with *Actinomyces necrophorus* have not been reported from Texas, and in the studies in Texas by Boughton and Hardy they were never observed. But in Wyoming and Colorado secondary necrophorus infections have been repeatedly observed, and reports from other countries indicate that such infections are relatively frequent. Marsh and Tunnicliff[3] have described an outbreak in lambs 3 to 4 weeks old in which the lesions on the mucosa of the mouth developed as a result of the activity of both *A. necrophorus* and the contagious ecthyma virus.

In mild attacks the eruption may be limited to a few papules at the commissures of the lips. As observed in the feed lots in Colorado the disease appears soon after arrival of the lambs and passes off in two or three weeks. Uncomplicated attacks in good weather cause no serious loss of weight and no mortality.

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VESICULAR STOMATITIS

(Sporadic Aphthae; Pseudo Foot-and-Mouth Disease)

Definition.—A contagious stomatitis occurring in equines and to some extent in cattle and swine; natural infection has not been observed in sheep. It is marked clinically by large vesicles that appear chiefly on the dorsal surface of the tongue. The virus is filterable and three types have been described.

Etiology.—*General Prevalence.*—Vesicular stomatitis has been observed occasionally in the United States in horses and cattle but it did not attract special attention until 1916, when it spread to thousands of horses in remount camps in the Middle West, was carried across the Atlantic, and described by French writers in horses originating in this country. Cattle were also affected but not extensively; a number showing vesicular stomatitis were found in a shipment in the Kansas City Stockyards in 1916, where the disease was thought by some to be foot-and-mouth disease. It has since been reported in cattle in Texas, Alabama, Indiana, and New Jersey. While vesicular stomatitis has not been reported as a natural disease of swine, in August 1943[1] a vesicular disease appeared in a Kansas City hog cholera serum plant in a group of animals that had been hyperimmunized twelve days before. It was identified by animal inoculation as the New Jersey type of vesicular stomatitis, and apparently it had been introduced through intravenous inoculation of the hypering virus. The disease caused severe local infection and even fatal septicemia.

The Virus.—In 1926 Olitsky, Traum, and Schoening[2] reported that the virus of vesicular stomatitis is filterable through Berkefeld V and N candles, through Seitz asbestos discs, and through Chamberland bougies, sizes L3 and L7. In guinea pigs, cattle, and swine, artificial injection was capable of producing lesions indistinguishable from those caused by the virus of foot-and-mouth disease. In cattle, however, the course was somewhat divergent, and intramuscular injection of the virus did not cause mouth lesions, while the virus of foot-and-mouth disease was positive in this respect. Horses were found to be very sensitive to oral inoculation, but were entirely resistant to the virus of foot-and-mouth disease. Similar results in respect to the virus of vesicular stomatitis were reported at the same time by Cotton.[3] In 1927 Cotton[4] reported further that the Indiana outbreak of May 1925 yielded a type of virus different from that found in the more severe New Jersey outbreak of September 1925; "this study has already yielded something of interest and that is the fact that there are two distinct strains of virus of vesicular stomatitis in the country, each of which will immunize

against itself, but neither of which will immunize against the other." Cotton[4,5] also reported in 1926 that the guinea pig is susceptible and more sensitive than large animals to the virus. The pathogenicity of the virus in other animals has been reported by Wagener,[6] who found it to be positive in swine, both by inoculation and contact exposure; by inoculation it was positive in wild rats and white rats, uncertain in rabbits, slight in cats, and negative in chickens and pigeons. In the horse and cow inoculation is most readily accomplished by placing fresh virus on a scarified area on the dorsal surface of the tongue; vesicles appear in from thirty-six to seventy-two hours. In these animals intravenous injection is positive, but subcutaneous is negative. Guinea pigs are inoculated by scarification of the metatarsal pad, and rubbing the virus into the surface with a small pledget of cotton; primary vesicles appear in from thirty to forty-eight hours. Inoculation experiments reported by the U. S. Bureau of Animal Industry indicate that sheep are not susceptible to the virus of vesicular stomatitis.

According to Mohler[7] natural infection appears to be entirely by direct contact, or from recently infected water troughs, feed troughs, bridles, or pails; it is not carried by caretakers, and the virus may leave the body before recovery is complete.

Symptoms.—The period of incubation is from two to five days. The initial symptoms are dullness, slight fever, anorexia, and salivation with strings of saliva hanging from the lips. Cattle may make smacking noises as in foot-and-mouth disease. These mild general symptoms are associated with or immediately followed by the typical eruption in the mouth. In the *horse* one first observes areas of redness on the tongue that are soon covered with vesicles from ½ to 2 inches in diameter. Within twenty-four hours the vesicles rupture, exposing a red granular surface that may be surrounded by a whitish fringe, a remnant of the covering of the vesicle. The exposed surfaces sometimes coalesce to form an area equal to the diameter of the tongue. Infrequently vesicles form on the lips and gums. The lesions are very superficial and healing occurs in from one to two weeks.

In *cows* there is a loss in milk and condition; the eruptions appear on the tongue, hard palate, lips and gums. In the unusually severe New Jersey outbreak, in addition to the mouth symptoms, a number of cattle developed lesions on the teats and feet. In describing this condition, Doctor Cotton[4] writes: "In a considerable number of the animals, lesions on the teats were the first to appear, to be followed later by vesicles in the mouth. . . . A large proportion of the cows had large vesicles or erosions on one or more teats, in quite a number, on all four of them. Many of the vesicles extended the full length of the teat,

and in several of them the entire tegument of the teat had sloughed away. . . . The lesions that appear on the feet of cattle through natural exposure consist of vesicles in the interdigital space usually extending its entire length, vesicles on bulbs of heel at junction of hair and hoof and also at points on the coronet." The foot lesions, heretofore described, unlike those of foot-and-mouth disease, are confined to one foot. An outbreak in cattle attacks chiefly the older animals, it includes not more than 50 per cent of the herd, and soon dies out. An attack confers immunity.

The symptoms of vesicular stomatitis in *swine*[8,9] are like those of foot-and-mouth disease.

Differential Diagnosis.—Compared with foot-and-mouth disease, it is less severe and less contagious; the feet and udders of cattle are not commonly attacked; sheep escape natural infection; it occurs chiefly in the horse, and vesicles appear on consecutive days. Diagnosis from the lesions is impossible after they are several days old, for the virus has disappeared.

Treatment.—Provide ground feed in the place of hay and an abundance of fresh water. Apply mild disinfectants, such as alum (3 per cent) or permanganate of potash (2 per cent). Segregate, and disinfect the premises.

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VESICULAR EXANTHEMA OF SWINE

Vesicular exanthema of swine is an acute highly contagious virus disease presenting lesions and symptoms identical with those of vesicular

stomatitis and foot-and-mouth disease in swine. But on inoculation the virus is negative to both cattle and guinea pigs, whereas the virus of foot-and-mouth disease and vesicular stomatitis is positive to both. The differential inoculation characteristics which identify it as an independent disease were observed by Traum,[1, 2, 3] who proposed in 1934 that it be named vesicular exanthema. It is not known to occur outside the United States.

Etiology.—The disease has been recognized only in California where it occurs chiefly in garbage-fed, occasionally in grain-fed, and in stockyard hogs imported for slaughter. It was first recognized in April, 1932,[4] when it was handled as foot-and-mouth disease although it failed to affect cattle, sheep, and goats. Two years later its real nature was confirmed, and control has since been limited to regulations imposed by the State. Since 1940 it has been constantly present. From December 1939 until June, 1940, the infection involved some 222,500 hogs, including one-fourth of all the hog premises in the state.[5, 6, 7] While the disease does not spread extensively, and may affect only a small number of hogs in a pen, once the virus gains access to susceptible hogs it may spread rapidly through the herd and in many instances the incidence of infection in a given group is 100 per cent. In the early acute stages the virus has been found in the blood and in the ham of a slaughtered hog. The chief mode of infection is thought to be through the medium of raw pork scraps in the garbage. Three or four immunological types have been demonstrated.

In June, 1952,[8] a serious outbreak of vasicular exanthema developed near Cheyenne, Wyoming, apparently from feeding raw garbage collected from a transcontinental train enroute from California. On June 11 it was found in Grand Island, Nebraska, in a herd of immune swine purchased in a garbage feed lot in Cheyenne. Within two months innumerable feed lots, nine serum plants, and several of the major stockyards had been quarantined, and within a year it had been found in approximately 40 states.

Symptoms.—It is an acute febrile disease in which confluent vesicles appear regularly on the soles, interdigital tissue, and the coronary region of the claws. Severe lameness may result from necrosis of the pododerm and sloughing of the soles and walls, especially in heavy hogs. Often vesicles also form on the snout, nose, and lips, on the mucous membranes of the mouth and nose, as well as on the udders and teats of nursing sows. As a rule the affected animals eat normally, but lesions may be so extensive that loss of weight results from inability to take food. The eruption is preceded and accompanied by a rise in temperature. Previous to rupture the vesicles are highly sensitive and the

more severe cases are found among the heavy mature individuals. In suckling pigs swelling of the nasal mucosa may cause fatal inspiratory dyspnea, and fatal secondary enteritis has been observed. While the course is short and the mortality low, there may be heavy financial damage from loss in condition, quarantine restrictions, and disruption of the normal routine. In mild outbreaks only a small percentage of the herd is affected; in severe attacks the morbidity may be high. The mortality is not over 5 per cent and deaths occur chiefly in suckling pigs. In some outbreaks there may be a predominance of snout lesions, while in others foot lesions will predominate, and lesions have been observed on the snouts of pigs only four days old. In certain years the eruption may be limited to the mucosa of the mouth or nose where vesicles and edema cause difficult breathing, and in sucklings asphyxiation. Losses result from condemnations, deaths of baby pigs, abortions, quarantine restrictions, and disruption of routine, and shrinkage may be so marked that thirty additional days of feeding time may be required before market weights are attained.

Diagnosis.—Since foot-and-mouth disease, vesicular stomatitis, and vesicular exanthema present identical symptoms and lesions in swine, special importance attaches to the diagnosis of any new outbreak of vesicular disease in this species. For a definite diagnosis in such cases animal inoculations are required. For test purposes cattle, swine, guinea pigs, and horses are used. For inoculation use only the coverings and lymph of unruptured or recently ruptured vesicles, since the virus loses its virulence rapidly. Inject cattle with an intradermic syringe in any of the mucous membranes of the mouth, or apply the virus to a scarified area. Inject a second animal in the vein or muscles; in cattle such injections are positive in foot-and-mouth disease, but negative in vesicular stomatitis. Cattle are negative to the virus of vesicular exanthema. Swine develop vesicular stomatitis, foot-and-mouth disease, or vesicular exanthema readily by intravenous or intramuscular injection, or by intradermal injection or by application of the virus to a scarified surface of the snout, mouth, or feet. In horses vesicular exanthema virus inoculated on the dorsal surface of the tongue causes mild lesions in about 50 per cent. The reaction consists of lifting the epithelium along the line of injection. No appreciable amount of fluid or only a small amount has been observed under the lifted epithelium. In vesicular stomatitis of the horse, in from 36 to 72 hours the tongue shows blanched and detachable epithelium at the site of inoculation which soon fills with clear, straw-colored fluid. These areas coalesce to form large vesicles containing from 5 to 10 cc. of fluid. In the next 24 hours the vesicle ruptures, leaving a deep, red, raw erosion, which heals slowly.

The following table shows the reactions to virus inoculations in the various species:

	Vesicular Exanthema	Vesicular Stomatitis	Foot-and-Mouth Disease
Swine	+	+	+
Horses	±	+	—
Cattle	—	+	+
Guinea pigs	—	+	+
Sheep	—	—	+

Control.—Since the heavy loss entailed by the spread of this disease in 1952-53, and the knowledge that it can be prevented by cooking garbage, a majority of the states have enacted laws which prohibit the feeding of raw garbage.

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FOOT-AND-MOUTH DISEASE

(Epizootic Aphthae; Aphthous Fever)

Definition.—An acute highly contagious vesicular eruption of the mouth, feet, and teats affecting all cloven-footed animals, but chiefly cattle, swine, sheep and goats. Equines are not affected. It is caused by an ultramicroscopic virus.

Etiology.—*Prevalence.*—Aphthous fever is constantly present in Europe, Asia, Japan, the Philippines, Africa, South and Central America. It has not been reported from New Zealand or Australia. Regardless of all precautions the disease appears frequently in Great Britain. Nine outbreaks have been reported in the United States: 1870, 1880, 1884, 1902, 1908, 1914, 1924 (2), 1929.[1,2] It was prevalent in Mexico from 1946 to 1952, and in Canada in 1952-53. It appeared again in Mexico in 1953. In countries where infection is permanent it often spreads to all susceptible animals within reach and then slumbers until more are

available. After each European war there has been a wave of the disease. Foot-and-mouth disease is the most contagious of animal plagues, and exposed animals are invariably attacked. The epidemiology is not affected by age, breed, climate, or season, except as these influences reduce the movement of animals.

Variations in Virulence.—Like most diseases caused by filterable viruses foot-and-mouth disease varies widely in intensity. Dr. Law[4] mentions a number of serious outbreaks, but states that they are infrequent. In 1918 a malignant form spread from Italy over Europe, causing a mortality of 50 per cent in some herds. In the United States, where a high degree of susceptibility exists, the disease has been relatively severe. In swine and sheep it is usually mild.

Immunity.—According to the report of the members of the Foot-and-Mouth Disease Commission,[5] "one may conclude that animals are, as a rule, wholly and solidly immune for a period of at least three months; that most of the animals lose their local resistance after seven months, but possess humoral immunity; and that eighteen months after infection practically all the animals have no local resistance, and a small number have no general immunity."

The Virus.—The virus is abundant in the milk and blood during the initial fever and in the vesicles that follow. Fresh contents of vesicles produce the disease in cattle in from one to three days when brought in contact with the abraded oral mucosa or if injected into the blood stream. Waldmann, Trautwein, and Pyl[6] have reported that the virus may be carried in the blood, and eliminated in the urine, for as long a time as 246 days after inoculation; thus the presence of foot-and-mouth disease carriers has been established. And Mohler[2a] states, "In one instance virus remained alive on the premises in California for 345 days." It is destroyed by pasteurization but is resistant to cold. Rats and rabbits may be inoculated, but not regularly. Horses are entirely negative to artificial inoculation. Guinea pigs are highly susceptible; inject intradermically into the hairless pad of the hind limb. Lesions appear at the point of injection in 24 hours, in the mouth in from 2 to 3 days, and the blood is infective within 24 to 48 hours. Hecke[7] reports the successful cultivation of foot-and-mouth disease virus. There are at least three types of virus: A, O, and C, and infection with one does not protect against the other. Strains of the Mexican virus[10] were identified as type A, and one strain was found to be type O. Blood and saliva may harbor the active virus before the lesions of the disease are present. Traum[5] reports that the activity of the virus in the vesicular fluid and in the coverings of both ruptured and unruptured vesicles decreases rather rapidly. Its presence was not demonstrated later than six days after the

appearance of the lesions. For the purpose of establishing diagnosis, only material from fresh vesicles not more than two days old should be used.

From the vesicles the virus is widely scattered by means of direct and indirect contact. The extensive outbreak in 1924 was traced to swine that received garbage from the Mare Island navy yard. Presumably this garbage was infected with supplies purchased in the Orient. A study of the 1914 outbreak gave only a plausible theory, namely, "The infection may have been introduced in merchandise brought to Niles, Michigan, from South America which found its way into a hog lot and in due time caused the development of the disease in hogs which were the first animals found infected" (Mohler).[1] In the course of the 1914 outbreak the following sources of infection were recognized: animals from infected stock yards, stables, or lots of local dealers, contact with neighboring herds at pasture, mating, railway cars, creameries—drinking infected milk, hog cholera virus and serum, persons—as visitors, workers, veterinarians, inspectors, stock buyers and peddlers, dogs, poultry, birds, public highways, contaminated streams and drinking water, garbage, and unknown. Wagener[8] states that "in England the wild rat has been found susceptible to foot-and-mouth disease, so that there can be no further doubt of the part played by rats in the spread of foot-and-mouth disease." Dr. Law[4] has reported that in 1902 the disease was produced experimentally in a calf by inoculation with cowpox vaccine having a Japanese label. In 1908 it was traced to contaminated smallpox vaccine (Mohler).[9] It was thought that sailors might have been the carriers in Texas,[20] in 1925. In slaughtered animals the onset of rigor mortis destroys any virus that may be in the muscles, but it retains its activity in the viscera, the lymph glands and the blood, even after freezing.

Morbid Anatomy.—In addition to the local lesions of the mouth, feet, and udder, congestion of the upper respiratory tract and hemorrhage in the epicardium are found. Punctiform hemorrhage may be present in the mucosa of the duodenum. Secondary infections involving the coronary region of the feet are not infrequent. Septicemic lesions are characteristic of rapidly fatal forms. One may find degeneration of the heart muscle, especially in young animals where the mortality is highest, and also in the malignant type.

Symptoms.—The period of incubation is from eighteen hours to three weeks; longer periods are explained by the virus being carried on the body before entering the tissues. With frequent exceptions the temperature rises from 2 to 3 degrees F. a few hours before the local symptoms are recognized. The initial mouth symptoms are those of an acute pain-

ful stomatitis: anorexia, abundant salivation, smacking of the tongue, grinding of the teeth, and redness of the oral mucosa. The distinctive eruption usually appears on the second day. In the mouth, vesicles and bullae are found. The smaller blisters are thin-walled and appear on the mucosa of the lips, cheeks, and dental pad; the larger are 1 to 2 inches in diameter, thick-walled, and appear on the dorsal surface of

Fig. 86.—Foot-and-mouth disease.

the tongue. These soon rupture, usually the same day, leaving a denuded, red, painful surface. The stomatitis is now more severe and anorexia may be complete. The saliva increases and drools freely from the closed lips. The denuded area heals in about a week, and uncomplicated cases recover in from ten to twenty days. Vesicles on the feet accompany or follow the eruption in the mouth. The initial symptoms are lameness, severe pain, heat, redness and swelling of the bulbs of the coronet. In one or two days vesicles appear in the interdigital space, especially at the anterior margin. Healing may be delayed by secondary infections which invade the deeper structures. Persistent recumbency and decubitis are common in cattle. On the udder, lesions appear shortly after the mouth eruption; the vesicles are confined largely to the teats and are ruptured by milking. Often the udder is swollen, the teat canal invaded, and the milk itself changed. All the members of the herd are attacked simultaneously, and the mouth vesicles all appear at the same time. Pain and anorexia cause rapid loss of flesh.

In *swine* the foot lesions are most prevalent, though vesicles may appear on the snout. The attack is milder than in cattle, and the resultant lameness may readily pass without an examination being made. As a rule the foot lesions first develop on the ventral surface of the claw at the anterior part of the bulb.

Differential Diagnosis.—*Vesicular stomatitis* closely resembles foot-and-mouth disease. In the United States vesicular stomatitis is rather infrequent in an enzootic form; it occurs in horses, cattle, and swine. An

extensive outbreak of vesicular stomatitis, affecting many remount horses and to a limited degree cattle, prevailed in the Middle West in 1917. At this time cattle in a shipment entering one of the stock yards were found to be affected with a form of stomatitis closely resembling, but not identical with, foot-and-mouth disease. Subsequent to 1917, similar incidents have been reported occasionally. In a discussion of the differential diagnosis of foot-and-mouth disease and vesicular stomatitis, Olitsky and coworkers[5] state, "In the doubtful cases, and especi-

Fig. 87.—Foot-and-mouth disease.

ally in the beginning of an outbreak, when a mistaken diagnosis in either direction has far-reaching effect, animal inoculations should be resorted to. It has been found that the virus of foot-and-mouth disease and vesicular stomatitis die rather quickly in the affected animals, so that to insure the virulence of the material to be tested, only lymph or the coverings of fresh vesicles should be used. This can be ground up in a sterile mortar with a small quantity of physiological saline solution. One or more susceptible cattle should be inoculated, intradermically, on the gum by syringe or by the application of the suspected material to the scarified area. One or more cattle should also be injected intramuscularly. By this latter method vesicular stomatitis has not, in the writers' experience, produced manifest lesions in cattle, while active foot-and-mouth virus has done so regularly. One or more horses should be exposed by applying the virus to a scarified area on the dorsal

surface of the tongue. Equines are very susceptible to vesicular stomatitis, but are very resistant to foot-and-mouth disease. The development or failure of development in the horse is the basis for differential diagnosis between foot-and-mouth disease and vesicular stomatitis." In the selection of test animals it is important to obtain individuals

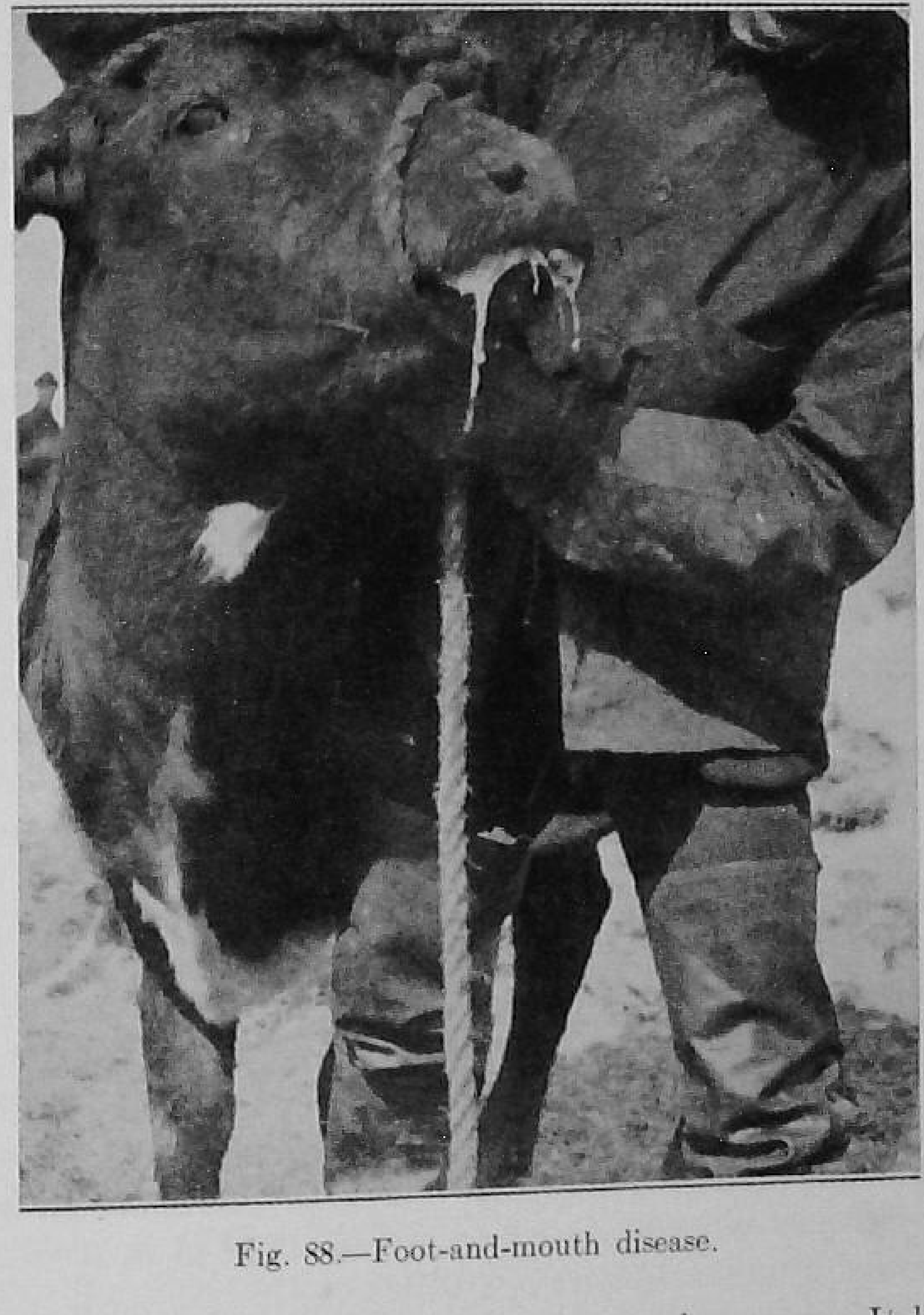

Fig. 88.—Foot-and-mouth disease.

that have been immunized by means of natural exposure. It has been observed that the eruption of foot-and-mouth disease occurs all at one time, within 12 to 14 hours, while that of vesicular stomatitis occurs in series over a period of a week. This variation has some value, but it is not entirely reliable.

In the recent outbreak of foot-and-mouth disease in Mexico, a rapid means of differential diagnosis of the two diseases was developed by means of differential typing of strains of field virus.[10]

Vesicular exanthema in swine presents symptoms and lesions identical with those of foot-and-mouth disease. In California, where it was confined until 1952, it was for a time diagnosed as foot-and-mouth disease for which owners were paid indemnity. In practice, any similar disease should be reported to state officials. And in more than one instance the official diagnosis in the beginning of an outbreak of foot-and-mouth disease has been wrong.

Lameness in swine and sheep should be investigated thoroughly. In these animals the feet are chiefly affected in foot-and-mouth disease, the symptoms are mild, and the disease may readily go undetected.

Aphthous (Mycotic) stomatitis affects only a few of the adult cattle in a herd, and, like other forms of nonvesicular stomatitis, it shows little resemblance to foot-and-mouth disease.

Prognosis.—Losses are heavy in all countries where the disease is permanently established. These result from diminished production, incomplete recovery, and quarantine restrictions, rather than loss of animals. Deaths are not infrequent, and in the malignant form the mortality may reach 50 per cent.

Control measures in the case of an outbreak in the United States are handled entirely by the Federal Bureau of Animal Industry. Under strict quarantine regulations, the disease is eradicated by means of slaughter and disinfection. An excellent description of regulations for the control of the disease in Europe is included in *Technical Bulletin* 76. In the 1946-52 outbreak in Mexico, vaccination was reported to be effective.

In 1938 Waldmann and Köbe[11] announced the discovery of a method of active immunization against foot-and-mouth disease. The vaccine is claimed to fulfill the following requirements: 1, inability to cause foot-and-mouth disease in cattle; 2, the production of a slight local and general reaction; and 3, the production of an adequate immunity. Immunity is evident from the fourth or fifth day and lasts for at least four to five months, according to present observations. Control by vaccination is employed in countries where the slaughter of infected and exposed animals is not economically possible.

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COITAL VESICULAR EXANTHEMA

(Genital Horse Pox; Eruptive Venereal Disease; Bläschenausschlag)

Definition.—Coital vesicular exanthema is a highly contagious vesicular eruption of the external genitals and adjacent skin. In the United States it is occasional in horses and rare in cattle; in Continental Europe it is common among cattle. Sheep and goats are rarely attacked. It is caused by a virus.

Etiology.—In those countries where the affection is frequent it is most prevalent during the breeding season. There is no explanation for its failure to become more prevalent in the United States. We have diagnosed it in cows in two or three herds in the ambulatory clinic in the past twenty years, and have had knowledge of at least one extensive and troublesome enzootic in cows in New York State. Possibly it occurs in cows in this country more often than reports indicate.

The virus is in the pustules or vesicles, as well as the secretions of the genitals, during the active stage of the disease. Infection takes place during coitus. The lesions excite females to estrum, and thus contribute to the transmission. Exceptionally, unbred heifers and foals are attacked; in such cases apparently the virus comes from the bedding or attendants. One attack does not confer immunity, and the infection does not spread from one species to another.

Reisinger and Reinmann[1] reported in 1928 that the cause is a filter-

able virus. Previous efforts by Zwick and Gminder[2] to establish this fact were without results; they were able, however, successfully to inoculate horses, sheep, and goats, from cows and to reproduce the lesions of granular venereal disease with the virus of vesicular vaginitis. But efforts to reproduce lesions of vesicular vaginitis from granular venereal disease were negative.

Symptoms.—*In cattle* the incubation period is from two to six days. Following artificial inoculation of cattle the symptoms appear in about twenty-four hours.

In cows there is usually a history of recent breeding to an infected bull. The initial symptoms are stamping the feet, restlessness, switching of the tail, arched back, stranguria, frequent urination, congestion and swelling of the vulva, and a purulent vaginal discharge. On examination the highly congested vaginal mucosa is seen to be thickly sprinkled with yellowish-white flat pustules from 1 to 3 mm. in diameter. Within one to two days these rupture and fuse to present the appearance of a fibrinous-like yellowish membrane. This membrane soon becomes detached, exposing superficial ulcers with irregular margins and a bleeding surface. Active suppuration occurs through a course of about two weeks when healing occurs without leaving nodules or scars.

In the male similar lesions may be found on the penis and prepuce.

In mares, there are swelling and congestion of the external genitals with the formation of papules, vesicles, and pustules. The symptoms and course are similar to those of the cow, but on healing the skin is left for several days or weeks with round, white, depigmented spots about ½ inch in diameter. Similar spots are left on the prepuce of the male.

Treatment consists in the application of a non-irritating astringent disinfectant, boric acid (4 per cent), chlorine solutions (1 to 2 per cent). An antiseptic of one-half per cent solution of equal parts copper sulfate and alum has been highly recommended. Dr. Williams[3] recommends an antiseptic wash composed of 1 ounce of carbolic acid with 2 ounces of tannin and 6 ounces of glycerine in 1 gallon of warm water.

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RINDERPEST

(Cattle Plague; Oriental Rinderpest)

Definition.—A highly contagious general disease of cattle characterized by a rapidly fatal febrile course, with localization of inflammation and necrosis on the mucous membranes, especially those of the digestive system. It is caused by a filterable virus.

History.—Rinderpest is of historical interest because of its leading position among cattle plagues from about the fifth century until the development of modern methods of disease control. Introduced from China into Western Asia with the invasion of the Mongols about the first century, it became permanently established around the Caspian and Black Seas. Since that date its record in Europe has been a series of plagues in which entire areas have been devastated of cattle, following each war, from the invasions of the Huns in the fifth century to those of Napoleon in the eighteenth. Its final eradication from Europe was in 1880, with the exception of a brief reappearance in Belgium in 1920. Dr. Law[1] writes that in 1841 all but a few Egyptian cattle perished within about two years after the plague was brought to Alexandria in a shipment of cattle from Roumania. According to Robles[2] it was introduced into the Philippines in 1886 in breeding cattle from French Indo-China, and it was believed to be finally eradicated in 1941. The disease is chiefly prevalent in Asia and Africa.

Etiology.—*Bacteriology.*—The virus was found to be filterable by Nicolle and Adel-Bey[3] in 1902. *Within the body* it is present in the blood, the tissue fluids, and all secretions and excretions of the diseased animal. It is found both during the incubation period and after recovery. It is said to be carried in the bodies of species of animals which resist an attack, though this opinion is disputed by some, and the influence of such possible carriers in the spread of the disease is insignificant.

The *method of infection* is chiefly by contact with diseased animals or their products, such as flesh and hides. It is improbable that the disease is commonly transmitted by intermediate carriers, such as food, water, utensils, attendants, cars, ships, etc. Infection occurs through the digestive tract, and the chief spread is along lines of cattle movements.

Outside the body the virus soon dies. It is destroyed by exposure to air, sunlight, putrefaction, temperature of 140° F., and weak disinfectants.

The young are most susceptible. From cattle it may be transmitted to sheep, goats, swine, and wild ruminants, though these animals are less susceptible than cattle.

Morbid Anatomy.—The most distinctive changes are found on the

mucosa of the mouth, the abomasum, and the small intestines. In the *mouth* the mucous membrane is congested. The cheeks and lips and ventral surface of the tongue are sprinkled with yellowish gray necrotic patches and erosion ulcers, and similar lesions may be found in the pharynx. In the *abomasum* the mucosa is highly congested and is sprinkled with small, flat caseous deposits and erosion ulcers. In the *small intestine* the mucosa is swollen, congested, and shows fibrinous and caseous deposits. Peyer's patches are infiltrated, swollen, and ulcerative. As a rule there are hemorrhages in the submucosa of the cecum.

Similar changes are found on the mucosa of the rectum, vagina, bladder, uterus, nasal passages, pharynx, larynx, trachea, and bronchi. Both the symptoms and the postmortem changes vary widely according to the breed, age, and nutrition, as well as the intensity of the attack. But the most characteristic and constant lesions are those found in the digestive tract.

Symptoms.—As described by Gomez,[4] "A rise in temperature always precedes the constitutional manifestations. Frequently, there is a premonitory period of nervous excitement which is more common in cattle than in carabaos. The animal is restless, rattles its chain, becomes vicious and may kick and gore. After the second and third day of rise of temperature, the animal becomes docile, and anorexia soon develops. It shows drooping head, lopping ears, congestion of facial veins, and general depression. Muscular tremors occur. Diarrhea develops after the third or fourth day of rise of temperature. From the second day of the symptoms, characteristic inflammatory changes of the mucous membrane appear. The conjunctiva becomes inflamed, the eyelids are swollen and tears run down from the inner canthus of the eyes which are at first watery and later mucopurulent. Photophobia is present in some instances and ulcerative keratitis has also been observed. There is a glary, transparent, tenaceous, and later, purulent nasal discharge. The nasal mucous membrane is reddened and later becomes spotted with petechiae. In the later stage of the disease the pulse is rapid and weak. The buccal mucous membrane is congested in spots, soon becoming covered with ecchymoses. Later, gray spots the size of a hemp or lentil seed appear. They become confluent and form pseudomembranes and when peeled they leave raw reddened ulcers. Saliva drools from the commissures of the mouth and often there is smacking of the jaw. Constipation is followed by straining and soon the feces become thin and a peculiarly fetid, watery diarrhea follows, and blood may be present. The mucous membrane of the rectum becomes swollen and congested. A cutaneous eruption may appear on the neck, behind the shoulders and

along the back. Pregnant animals may abort. Soon the animal becomes weak and death occurs in from three to ten days."

The most distinctive symptoms are the high continuous fever, erosions in the mouth with grayish yellow deposits, severe intestinal attack, and rapid emaciation. It may be confused with malignant head catarrh, which is sporadic, foot-and-mouth disease, and coccidiosis. In coccidiosis, examination of the feces reveals the parasite and the disease cannot be reproduced by inoculation.

In the *eradication of rinderpest,* immunization with *tissue vaccine* has proved to be effective against the spread of the disease. This was first prepared by Boynton[5] and used extensively in 1922 in the Philippines. The original vaccine was a mixture of ground tissues, glycerin, and phenol, attenuated by heat. Later the tissue vaccine was modified, and its use simplified by Kelser,[6] who added chloroform to kill the virus. A single injection of the chloroform-killed tissue vaccine produces immunity—Rodier.[7] In 1932 Robles[2] reported that "the improved chloroform-treated vaccine is capable of giving a high degree of protection to about 70 per cent of vaccinated carabaos, and 90 per cent of cattle. . . . Vaccinated animals are adequately immune to artificial infection for a period of at least three months." The vaccine was further modified to form a dried powder which could be widely distributed without refrigeration. In the summer of 1941 the Philippine Islands were declared free from rinderpest.

In India goat-tissue virus is used extensively in immunization against rinderpest. It is inexpensive and is said to be highly effective. In an editorial comment in the *Veterinary Record* for Jan. 14, 1939, on its use in India the following statement is made: "It shows that a grave and large-scale problem in disease control which had apparently baffled all attempts to provide a really satisfactory solution may prove, as the result of systematic research, to be unexpectedly and surprisingly easy of solution." According to D'Costa[8] the use of goat-tissue virus in India is an important and useful step. Later reports[9] reveal a marked increase in the use of goat-tissue vaccine in India, where it is employed for prophylaxis and in actual outbreaks. Serum is sometimes given with vaccine to check reactions, and goat-blood virus is also used. Yet one reads in 1950 that rinderpest is first in order of importance in India, where regardless of effective serums and vaccines 93 per cent of the cattle are allowed to face outbreaks without any protection, and one of the causes of the chronic indebtedness of the Indian farmer is rinderpest.[10]

Because of the failure of virus to survive for any length of time outside the body, the control of rinderpest is relatively easy except where livestock is maintained under primitive conditions.

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CONTAGIOUS PLEUROPNEUMONIA OF CATTLE

(Lung Plague; Lungenseuche)

Definition.—A contagious disease of the lungs of cattle in which there is a marked exudation into the intralobular connective tissue, diffuse pneumonia, and serofibrinous pleuritis. It is caused by a filterable virus.

History.—While at the present time the chief distribution of lung plague is in Africa, Australia, and Asia, it has been one of the more important cattle plagues of history. This was especially true in the nineteenth century in connection with exportation of cattle from Central Europe to the Scandinavian countries, England, Ireland, America, Australia, and Africa. In the United States the disease was introduced in 1843 and eradication was not complete until 1892. Europe was free from infection for many years previous to the World War, when it again entered with cattle originating in Russia or Roumania; it was soon eradicated, however, and its presence in Europe is now limited to the eastern part.

Etiology.—The active agent is described as a pleomorphic microorganism that may be seen at a magnification of from 1500 to 2000 as opaque points, vibrios, and branched forms (*Asterococcus mycoides*). It has been cultured by Nocard and Roux. *Within the body* the virus is carried in active and latent cases, and in recovered animals. In affected animals it is present in the urine, milk, and uterine exudate at the time of parturition. The *method of infection* is by means of direct contact with sick or convalescent animals. The virus may be carried

indirectly by people, small ruminants, dogs, cats, in milk, excretions, etc. Transmission occurs most readily by direct contact between acute cases and susceptible cattle. *Outside the body* the virus retains virulence for a long period; it has withstood drying for more than 100 days.

The species affected, in addition to cattle, are: buffalo, bison, yak, sheep, and goats.

Morbid Anatomy.—Lung plague is a primary bronchopneumonia with marked involvement of the interlobular connective tissue and pleura. As in other forms of pneumonia in bovines, the lesions may become chronic.

In the *acute* form the chest cavity usually contains turbid fluid and the pleura is covered with fibrinous exudate. Infiltration of the interlobular connective tissue gives to the lung a marbled appearance, and on cut section this is especially striking. In addition, the cut surface presents extensive grayish or reddish areas of consolidation—lobar pneumonia. The bronchial and mediastinal lymph glands are swollen.

In the less frequent *chronic* form, adhesive pleuritis may be found on removal of the chest wall. The lungs contain necrotic foci with connective tissue walls, areas of chronic pneumonia, and caseation or calcification.

Symptoms.—The onset is gradual. For the first two or three weeks a cough and fever are the chief signs. During this early, chronic stage of development the penumonia is lobular and the progress slow; even auscultation and percussion are negative. After the lung lesions are sufficiently advanced, symptoms of acute bronchopneumonia are prominent. The animal stops eating, rumination and milk flow cease, and symptoms of intense pleuropneumonia appear. These are high fever (104° to 106° F.), dyspnea, mucopurulent nasal discharge, sometimes tinged with blood, painful cough, marked pain on intercostal pressure, dullness on percussion, and a wide variety of râles. The immediate mortality is estimated at from 30 to 50 per cent, with incomplete recovery in an additional 30 per cent. Death may occur at the end of a week after the onset of the acute stage, but more often it occurs at the end of two to four weeks. In older animals the course tends to be chronic, and the symptoms are similar to those of advanced pulmonary tuberculosis.

Diagnosis.—Because of the similarity of the pulmonary lesions, lung plague may be confused with the pulmonary form of hemorrhagic septicemia. Other forms of pneumonia lack the characteristic interlobular thickening, and are usually noncontagious.

According to Ziegler,[1] a positive diagnosis can be made by means of a histological examination of the interstitial and peribronchial tissue. Around the blood vessels of the bronchi, there is a light inner zone con-

sisting of reticular tissue containing a few lymphocytes and a dark outer zone consisting of degenerating white blood corpuscles. These perivascular foci are held to be pathognomonic. Calves may be inoculated with filtered pulmonary exudate.

Control.—The most effective procedure is to slaughter all infected and exposed animals, and disinfect or destroy contaminated premises. While it is possible to immunize against the disease, such protection does not extend to all of the vaccinated animals. Purchase[2] has written an extensive report on vaccination with "culture-virus" in Kenya Colony.

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LOUPING ILL

(Trembles, Encephalomyelitis of Sheep)

Definition.—Louping ill is an enzootic encephalomyelitis of sheep in Scotland and northern England caused by a neurotrophic filterable virus and characterized by nervous symptoms, such as hyperesthesia, and motor irritation.

While it has been recognized as a serious scourge for many years, and the tick, *Ixodes ricinus*, has long been suspected of being concerned in the transmission, exact knowledge of the disease has only recently been acquired through researches conducted by various workers in Scotland.[1, 2, 3, 4, 5] They have reported that the disease can regularly be transmitted to sheep, pigs and mice by the intracerebral inoculation of material from the brain and spinal cord to sheep, that the cause of the disease is a filterable virus, and an active immunity can be produced by an intradermic or subcutaneous injection of the virus, and that under natural conditions the disease is transmitted by ticks (*Ixodes ricinus*).

Etiology.—Louping ill occurs chiefly as an endemic in spring and early summer in lambs and yearlings on pastures of certain hill farms. In permanently infected flocks there is an age immunity attributed to a mild attack in early life. But if sheep are brought from a locality in which louping ill does not occur into a louping-ill district, a large proportion are liable to develop the disease.

Sheep, monkeys, pigs, cattle and mice are susceptible to intravertebral, intraspinal and subcutaneous inoculation of brain emulsions from affected sheep, and from these animals it may be retransmitted by intracerebral inoculation. Under natural conditions, sheep, cattle and prob-

ably pigs are susceptible (Brownlee).[8] Infection can be transmitted by ticks that have been engorged upon cases of louping ill and subsequently engorged upon healthy sheep; it can also be transmitted by blood drawn from an affected animal during the febrile attack, but the only reliable method of producing a typical case of louping ill in sheep is by introducing the virus directly into the nervous system. Brownlee writes that although the virus of louping ill attacks typically the nervous system, it does not do so at once. Before massing its attack on the nervous system, it first multiplies in the blood, and it also enters the lymph glands and the spleen. In from two to three days after intracerebral inoculation there develops a fever which reaches its height about the fourth or fifth day and then rapidly declines. With the drop in temperature there is a disappearance of the virus in the blood. In those cases in which the febrile reaction is followed by symptoms referable to nervous disturbance, these appear on the fifth or sixth day, and death usually occurs on the sixth or seventh day after inoculation. There is no evidence that the disease spreads by contact. Subcutaneous inoculation of formalinized vaccine prepared from the brain and spinal cord of sheep produces antibodies capable of neutralizing virus introduced into the general circulation, thus preventing the active agent from entering the brain and spinal cord. Gordon[7] reports that in considering methods of prophylactic vaccination for the control of louping ill it was found that the central nervous system is a difficult tissue to immunize. "Subcutaneous injection of living virus, when followed by a febrile reaction in the inoculated animal, immunizes the central nervous system, but this method is attended by a risk of setting up the disease." Experimental immunity persists for at least eleven months.

It was also observed that a febrile reaction can be experimentally produced in sheep by infesting them with ticks collected from infected pastures, and also by the serial inoculation of sheep with blood obtained from these tick-infested sheep; but the reaction did not protect against the subsequent inoculation of louping-ill virus. This other malady is termed tick-borne fever.

Morbid Anatomy.—Gross lesions are absent. In a description of the histopathology of louping ill Brownlee and Wilson[6] record that a varying degree of destruction of the Purkinje cells of the cerebellum of both natural and experimental cases of the disease was constantly met with; that severe damage to the nerve cells of the medulla and all parts of the spinal cord, though not constantly present, often occurred in both natural and experimental cases; that in the pig nervous tissues show very intense cellular infiltration and relatively little nerve cell destruction; and that in the mouse the principal lesion found was necrosis of

the majority of the large nerve cells of the medulla and cord. No lesions were found in cases clinically resembling louping ill, but from which no virus was obtained.

Symptoms.—The period of incubation is from six to eighteen days. On infected farms a large proportion of the old sheep are immune and 20 per cent of the yearlings from such farms may be immune. The disease begins with dullness, and a temperature reaction, 106°F., often with no other signs of illness. These are followed by hyperesthesia, motor irritation, and deranged consciousness in the form of fright and excitement when approached. When handled the sheep trembles and develops clonic spasms with the head drawn back or sidewise. There may be salivation, champing of the jaws and rolling of the eyes. The leg movements may be stiff or jerking, resembling stringhalt, and leading to paralysis of one or more limbs. Within a few hours or a day or two the animal goes down and soon dies. Pool writes that there is a great deal of variation in the different stages of the illness, and animals which were apparently in normal health at night were frequently comatose or even dead the following morning. Sometimes the animals die without showing any symptoms of disease of the nervous system. Where tick infestation is heavy it has a marked debilitating effect. The mortality is high. The total loss is estimated by Stockman at from 2 to 3 per cent.

A vaccine prepared from formalinized brain and spinal cord and spleen emulsions has been tested on a large scale with very encouraging results—Brownlee, Gordon. Experimental vaccination at the Moredon Institute in Scotland indicated that 10 cc. of a 1:1000 powdered brain emulsion was a safe and effective immunizing agent, but in field use it transmitted louping ill to many sheep with fatal results.

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SCRAPIE

Definition.—Scrapie in sheep is a chronic virus meningo-encephalitis that begins with trembling of the lips, ears, and limbs, followed by intense pruritus, emaciation, and finally death after a prolonged course.

Etiology.—For many years scrapie has been known in Britain. In 1938 Schofield[1] described a case of scrapie in Canada in a 3-year-old ewe that had been imported from Scotland, and in 1945 it was reported that "in the last six years scrapie has been diagnosed in three Canadian flocks. In the United States it was first seen in California in October, 1952;[2] and in 1953 it was reported in five flocks in Ohio and one in Illinois. In 1950 Wilson, Anderson and Smith[3] reported experimental transmission of the disease by intracerebral injection of a filtered bacteria-free emulsion of a diseased brain, medulla and spinal cord. The incubation period was four to five months, and the course averaged 7.7 weeks. The virus remained viable for months when dry. That the disease may be acquired at pasture, without direct contact, was shown in an experiment by Greig[4] in which normal and diseased sheep exchanged pastures twice weekly without direct contact. After thirty-nine months of such exposure scrapie appeared in one of the normal sheep, and within the following three months nine other contact ewes developed the disease.

Morbid Anatomy.—Large vacuoles in the nerve cells of the medulla and spinal cords are the only constant abnormality.

Symptoms.—There is a striking similarity in the symptoms. In natural infection the onset is insidious in the form of fine tremors over the head and neck which cause slight nodding movements. The head and neck are held high and stiff and the expression is staring or fixed. Excitement causes muscular tremors to appear over the thighs and flanks, and physical restraint may cause convulsions. Because of the pruritus affected sheep are constantly biting or nibbling at almost any part of their bodies; they rub their sides and necks against fences until the body is denuded of large areas of wool, and these areas are hypersensitive to the slightest touch. There may be thirst and grinding of the teeth. Emaciation develops gradually and becomes extreme. The course is reported at six weeks to six months and is said to be invariably fatal. The rate of incidence in a herd varies widely and is commonly reported to be between 4 and 20 per cent. In a herd there may be only an occasional case over a period of several years, or as high as 50 per cent may be affected.

Control.—The only successful method of control has been complete slaughter of the affected herd, and this has not always proved to be

effective. Because of the insidious character of the disease, the long period of incubation, and the possibility of pasture exposure, disposal of all affected and in-contact animals is probably the most logical procedure.

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BLUETONGUE

Definition.—Bluetongue is an acute stomatitis of sheep caused by a virus that is transmitted by biting insects. In a severe form there is general involvement with localization in the digestive and respiratory systems, the muscles, and the hooves.

Etiology.—As a major problem it is of chief importance in South Africa, where it has been prevalent for at least fifty years. The disease was reported in Cyprus in 1924,[1] in Turkey in 1945,[2] and in Israel in 1951.[3] In 1949 Gambles[4] wrote that until 1943, when bluetongue was recognized in Cyprus and Palestine, it had not been observed outside of Africa. In 1952 it was described in Texas by Hardy and Price[5] as "sore-muzzle of sheep" that first appeared in 1948 and had not been previously described in the United States. No cases were observed in 1949 and 1950, but in June 1951 it was again reported and in four weeks it had been identified on 20 ranches; it was both sporadic and enzootic in elevations up to 2000 feet, and in animals that had not been on pasture in ten months. In 1952 it appeared in California,[6] where by November it had spread to all sheep-raising areas, affecting 325,000 sheep with a mortality of 5 per cent. The cause was demonstrated by McKercher et al.[7] to be a virus that was identified by Alexander of South Africa as a mild strain of bluetongue virus. The disease again was quite prevalent in 1953, and has been diagnosed in Arizona, Nevada, and Utah.[8] It is a seasonal disease that has been transmitted experimentally by the injection of Culicoides,[9] and it is believed to be transmitted by these biting insects. In South Africa eleven strains of virus have been identified while only two or three strains have been identified in this country.[8]

Morbid Anatomy.—There is hyperemia of the entire oral and nasal mucosa, ulcers on the dental pad, the tip of the tongue and immediately posterior to the incisor teeth. The nostrils, muzzle and nasal septum

are inflamed. Small scattered areas of degeneration of the muscles are present throughout the body, with large hemorrhages at the base of the pulmonary artery and aorta. The submaxillary region and lungs are edematous; the body cavities and the pericardial sac contain fluid. Other lesions are enteritis, inflammation of the coronary region seen as a red zone or band on white-footed animals. The cadaver is emaciated.

Symptoms.—Both a severe and a mild form have been described; in the severe form the incubation period is given as three to seven days. Sucklings are less susceptible than adults and mature rams are said to be most susceptible. The first symptom is an edema of the lips and a serous nasal discharge which soon becomes purulent and adheres to the sensitive and hyperemic underlying skin. Depression, salivation, and rapid loss of weight appear early. The temperature is from 102° to 104.5° F. Ulcers appear on the dental pad and the tip of the tongue and diarrhea is frequent. Lameness occurs but is not constant. The course is from three days to three weeks, depending on the severity, and the estimated mortality is from 5 to 10 per cent, but may be much higher. The morbidity is less than 30 per cent with an average of 10 per cent. Near the end of fatal cases there are râles, frothy nasal discharge, and open-mouth breathing with death in from one to six days due to secondary pneumonia, weakness and emaciation; recovered animals remain poor.

Control.—The disease has been prevented by injection of an egg-embryo vaccine made from strains of virus obtained from an area where the disease exists, but not from distant areas. Such a vaccine has been used successfully in South Africa, in Cyprus, and in Turkey.

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CHRONIC INFECTIOUS DISEASES

TUBERCULOSIS

Definition.—A chronic infectious disease caused by *Mycobacterium tuberculosis* and characterized by the development of nodules of granulation tissue, or tubercles, which undergo calcification, caseation, or abscess formation. Primarily it affects the lymph glands. Tuberculosis varies widely according to the organs involved and the degree of resistance of the individual. Since the fourth century B.C. it has been regarded by many as infectious, and this opinion was confirmed in 1867 by Villemin.[1] In 1882 Koch[2] discovered the tubercle bacillus, and in 1890 tuberculin.

General Prevalence.—In the dairy districts of Europe and England the percentage of tuberculosis in cattle is so high that no serious attempt towards eradication has been made. Control measures have been restricted to the elimination of advanced physical cases and the raising of calves in such a manner that infection may be avoided. In the United States in general the percentage of bovine tuberculosis is low, but some of the older dairy sections have had from 50 to 100 per cent. In the United States it is officially estimated that it dropped from 4.9 per cent in 1918 to 0.14 per cent in 1951. While all species of domestic animals are attacked, it is of chief economic importance in cattle, swine, and poultry. Tuberculosis in poultry is most prevalent in the Mid-Western and North Central States.

Etiology.—The tubercle bacillus is a slender rod 2 to 4 microns long; it takes a characteristic stain, is "acid fast," and in cultures gives a distinctive growth. According to the species three types are recognized, the *bovine*, the *avian*, and the *human*. As in all specific infections the virulence of the individual strains varies.

The *bovine* type is capable of producing tuberculosis in practically all mammals. In experimental animals (rabbits and guinea pigs) it exceeds all others in virulence. With the exception of the human type, it is the only form of any importance in man, where it causes tuberculosis of the joints and lymph glands of children fed on infected cow's milk, and studies by Griffith and Munro[3] have shown that in Scotland 160 of 2,769 cases of pulmonary tuberculosis were of bovine origin, with evidence that the digestive tract was the channel of entry. For man the bovine type is as virulent as the human type. It is the chief cause of frequent extensive tuberculosis in swine, of the occasional case in the horse, and of the rare case in sheep and goats. With the exception of parrots, birds are not susceptible.

The *avian* type is responsible for a large majority of the slight cases of tuberculosis found in swine, and according to a 1937 Federal Report they are capable of causing "progressive generalized tuberculosis in a small percentage of swine affected with this organism." Fowls are highly susceptible. In the United States the avian type has only rarely been reported as pathogenic to cattle. Schalk[4] found that 75 per cent of cattle exposed to avian infection became sensitized to avian germs and reacted to avian tuberculin for a period of two to five months. Of 507 cattle tested with avian tuberculin in Fargo, North Dakota, 15.5 per cent reacted, while only two reacted to bovine tuberculin. He concluded that avian tuberculosis is not an economic problem in cattle. In Denmark it is regarded by Plum[5] as the cause of more than 2 per cent of bovine abortion. It is pathogenic to the rabbit but not to the guinea pig. Avian tuberculosis in sheep has been reported by Harshfield and Roderick,[6] and the avian type of tubercle bacillus has been reported to be a cause of reaction to the tuberculin test in calves—Federal Report, 1938, p. 63.

The *human* type is not naturally pathogenic to domestic animals, fowls, or rabbits. Guinea pigs are susceptible to infection.

In the body tubercle bacilli multiply and are found in all of the lesions and in all body cavities with which they communicate. Koch believed that the high prevalence of pulmonary tuberculosis proved an equally high infection by inhalation. Numerous experiments have proved, however, that regardless of the source, the tubercle bacilli enter domestic animals almost entirely by ingestion. They pass to the intestine, enter the circulation, and are filtered out by those organs and tissues in which the soil favors colonization and growth—lymph glands, lungs, serous membranes, genitals, etc. Evidence in support of ingestion is found in the fact that a cow with advanced pulmonary tuberculosis may occupy a stall between susceptible individuals for a long time and not spread the infection, if individual mangers are used. I have observed numerous instances of this kind in northern New England in stables of wooden construction, in which the mangers were separated by tight board partitions. Tubercle bacilli are eliminated from lung lesions in enormous numbers, and from here they constantly pass to all parts of the alimentary tract. Samples of mucus taken from the esophagus with a sputum cup may contain organisms regularly, even from a cow containing a single small lung lesion. Tubercle bacilli are also present in the feces of such an animal. In a similar manner they escape from an infected uterus into the vagina and its discharges. Thus they constantly enter the gutter, the manger, or the water trough from either a small or a large tuberculous lesion. According to Watson,[7]

"it has been proved, over and over again, that tubercle bacilli may be secreted from the milk from the udder in which neither manifest tuberculosis nor microscopical evidence of tuberculous lesions are present. . . . Many such cases have been reported in the literature and in some of these the tuberculous lesions discoverable at postmorten examination proved to be very slight in extent and limited to one or more of the lymphatic glands. . . . We have determined that cattle may harbor and excrete virulent tubercle bacilli for long periods of time and, at postmortem examination, show little evidence of tuberculosis. . . . It frequently happens that virulent tubercle bacilli are present in groups of lymph glands which appear to be quite normal—N. V. L. (no visible lesion) glands—their presence being determined by biological tests on guinea pigs. The observations and experimental data that are being accumulated point to considerable resistance to active disease in this category of cases."

Outside the body the habitat of the tubercle bacillus is in mangers and feeding troughs, as well as in buckets and water troughs not supplied with constant circulation. There is little evidence of contamination of water troughs through which water constantly flows freely, as from springs. But when the water is removed only by drinking, bacilli are constantly being added and infection becomes massive. This is also true of water holes. Traum[8] has reported finding infective bacilli in water holes 687 days after exposure. He concluded that environs contaminated with tuberculous discharges from cattle become free from infective material soon after the media in which they are contained are freed from sensible moisture. Infection of pastures soon dies from exposure to sunlight. Milk from tuberculous herds is practically always infected, while skim milk and buttermilk from creameries carries the infection, even after it has been "pasteurized." Such milk may not be properly heated. There is little or no evidence of the presence of bovine tubercle bacilli in sufficient numbers to cause the disease, in the air or dust of stables. The common watering trough or hole, common manger, and milk are the chief habitat outside the body.

Predisposition.—The effects of age, climate, nutrition, sunlight, individual predisposition, and heredity have little influence. If cattle daily swallow tubercle bacilli from the common sources of infection, as mangers, troughs, water holes, water troughs, and milk, the number that escape disease is insignificant. Tuberculosis is just as prevalent, and more difficult to control, in sections of California where cattle never enter barns, as it is in Vermont where they are housed more than half the year.

Methods of Feeding and Watering.—It is believed by some that lack

of light and cleanliness in stables is the chief cause of the spread of infection among cows. But of far greater importance is the construction of the mangers and the method of watering. Especially to be condemned is the watering of stock in a common concrete manger, if the water enters slowly and is allowed to stand. No more effective means of spread can be devised, if a member of the unit is a spreader. Equally dangerous is the watering of stock from a trough into which water enters and from which it is not removed except by drinking. An entire herd may become infected in this manner in a single summer, if one of its members has an open lung lesion. Disastrous results have followed the use of individual drinking cups when so constructed that sediment from a cup can gravitate to the main water pipe. Feeding in a common manger is less dangerous, but this practice has caused the loss of many animals, and led to the construction of individual mangers, or a manger shared by only two or three cows. It is a common observation that tuberculosis spreads more rapidly in a modern concrete stable, where cows are fed and watered in a common manger, than it ever did in old-fashioned stables of wooden construction with individual mangers where each cow always occupied the same stall. *Milk and dairy by-products* are a common source of infection in calves. An outbreak of tuberculous mastitis due to udder irrigation with contaminated apparatus has been reported by McFarlane,[9] and cases of infection of cattle with the bovine type from man have been described by Tice.[10]

Purchases.—The high prevalence of tuberculosis in one area and comparative freedom from it in another may be explained by the methods of obtaining replacements. Where these are raised on the owner's farm, and not purchased through cattle dealers, the danger of introduction of infection is greatly reduced. Where additions are entirely by purchase, and trade in cows is active, escape from infection is difficult, even when the purchased cows are required to pass a tuberculin test. In recent years, however, since the establishment of tuberculosis-free areas, and many accredited herds, cows bought after passing a tuberculin test have less frequently proved to be tuberculous.

Pathology.—*(a) The Tubercle.*—Osler[11] states that "a 'tubercle' presents in its early formation nothing distinctive or peculiar, either in its components or in their arrangement." *In its development* the tubercle bacilli multiply; the fixed cells multiply; and form epithelioid and giant cells; polynuclear cells assemble, but soon perish; and an outer zone of fibrous tissue forms. *In its degeneration* central necrosis with caseation and calcification destroy, while the formation of an outer zone of fibrous tissue (sclerosis) forms a protective barrier. Individual tubercles are microscopic; they fuse to form *miliary tubercles* the size

of a millet seed, and these unite to form tuberculous masses.

Through rapidity of growth and failure of the protective wall, vital organs are invaded and their functions destroyed. In the lungs and occasionally in the retropharyngeal lymph glands, there is a tendency to abscess formation. While the varations in this process are endless, there are three chief reactions from infected individuals: 1. The bacilli are promptly destroyed on entering the body. 2. After a certain development the tubercle is walled off and growth is arrested. 3. The development proceeds either slowly or rapidly without interruption until the victim is destroyed.

(b) Distribution of the Tubercle.—Postmortem examination is usually made in routine abattoir inspection, or because of reaction to the tuberculin test. It is infrequent that a case of bovine tuberculosis is allowed to progress until death from the disease occurs. The *lymph glands* are the primary seat of infection, involvement of the lungs and other organs being secondary. While lesions may develop in any part of the lymphatic system, they occur chiefly in certain groups of glands: retropharyngeal, bronchial, mediastinal, and less frequently, in the mesenteric or portal. The size of the tuberculous growth may vary from that of a hardly perceptible calcareous spot to a calcified mass weighing several pounds.

Pulmonary tuberculosis is second in frequency to the glandular form. Small tubercles are most readily recognized by firmly palpating the uncut lung between the thumb and fingers; they may be found in any part of the lung, but are most common in the larger lobes. If the bronchi are carefully and extensively slit, one may find a tenacious bronchial exudate, and a direct large or minute communication between a bronchus and a degenerating tuberculous mass; this is termed an "open" lesion. A distinction is made between an "open" and a "closed" lung lesion, but I have never been able to learn how to make the distinction. The parietal pleura may show recent, almost imperceptible, proliferation, or extensive growths—"Pearl disease."

The *peritoneum* occasionally is uniformly and extensively covered, especially over the omentum and viscera, with small, dark yellow spots which on histological examination prove to be tubercles; in a more advanced stage characteristic tuberculous growths are present. This form is usually associated with greatly enlarged tuberculous oviducts, and, sometimes, with tuberculosis of the uterine mucosa; the mucous surface is calcareous and the cavity contains sand-like calculi.

Skin tuberculosis.—The subcutaneous lymphatic vessels of the limbs sometimes show thickenings in the form of nodules or cords; these are found in the region of the shoulder, the tarsus and the fetlocks. They

contain acid-fast rods that have not yet met the specifications required of the tubercle bacillus, since they do not grow in cultures and are not pathogenic to guinea pigs. They seem to cause reaction to tuberculin and are officially termed "skin tuberculosis." Mohler[12] has reported that "a number of so-called skin lesions have been obtained from various sections of the United States in an attempt to culture the organism causing this condition and to transmit the virus to laboratory animals. All attempts gave negative results." According to Hastings,[13] "the preponderance of evidence collected by a number of workers is against the skin lesions being tuberculous."

Other organs in which lesions are not rare are: the liver, testicles, udder, spleen, kidneys, cerebral meninges, joints (stifle), and intestinal mucosa. When the lesions are restricted to a limited area the disease is said to be *local;* when infection is carried in the general circulation and tubercles develop in many areas, it is termed *generalized.*

Symptoms.—Because of wide variations in the distribution of tubercles in the body, bovine tuberculosis presents a wide variety of symptoms. With few exceptions the course is chronic. The history of the herd, or the presence of purchased additions is often helpful in making a diagnosis. In sections where the rate of tuberculosis is high any chronic disease is suggestive of this infection.

Poor condition is the most frequent indication of tuberculosis. This appears if the disease is extensive, or if it disturbs the respiratory, digestive, or circulatory apparatus. It should be judged in relation to the average nutrition in the herd, the age, and other debilitating diseases, such as metritis, traumatic gastritis, chronic bowel catarrh, painful conditions in the feet or joints, and Johne's disease. The temperature is usually normal.

The respiratory system is the most frequent seat of clinical tuberculosis in cattle. Dyspnea and increased frequency of breathing are often present in pulmonary and generalized forms. Swelling of the retropharyngeal lymph glands causes compression stenosis of the pharynx with inspiratory dyspnea. Inspiratory dyspnea in a cow is nearly always the result of tuberculosis of the retropharyngeal glands. Pulmonary lesions often lead to cough. At first this may be harsh and dry, but usually it becomes soft, moist, and low. When one remains in the stable with such an animal, a cough will be heard at irregular intervals regardless of the time of feeding or drinking, though it is most marked in the morning. Stable men may report that such an animal has been affected with a "cold" for weeks. Pinching the trachea may induce a cough, and this is one of the common symptoms of advanced lung lesions; it is especially significant when weak, moist, and low, and when

it can be easily and repeatedly produced. It is the most frequent sign of a lung lesion. Auscultation may show that one lung gives a louder vesicular murmur than the other. This sound has such a wide normal variation that deception easily occurs. Rapid brief exercise may induce sounds that do not otherwise exist. The presence of râles is positive evidence of disease, but a lung may have well-marked open lesions and still not reveal a cough or abnormal breathing sounds on only one examination. Abnormal respiratory sounds may also rise from other causes, such as emphysema in old animals. A tuberculous cow may give marked râles one day and none the next. Percussion of the thorax may reveal an area of dullness, pain, or induced cough; the latter is of special significance. A sample of sputum taken from the esophagus and injected into a guinea pig may reveal open and even unsuspected cases.

The Udder.—In the vast majority of cases of open tuberculosis the lesions are either in the udder or the lungs. In suspected cases milk should be injected into guinea pigs. Following parturition, lesions in the udder have a tendency, as in other organs, to increase in growth. According to Hess tuberculosis usually attacks the left hind quarter and rarely attacks both hind quarters at the same time. Clinical tuberculosis of the udder usually is associated with a swelling of the supramammary lymph glands, and this swelling may occur even when the disease is confined to an anterior quarter. The local symptoms are in the form of nodular, circumscribed, or diffuse swellings that develop slowly without heat or pain and are not unlike those found in chronic streptococcic mastitis. The milk from the affected quarter may be slightly diminished at first, but the character is unchanged; after a few weeks it may become thin and watery.

The Lymph Glands.—Because of their location, the superficial lymph glands are of great value in the diagnosis of tuberculosis. Disease of these glands seems to be frequent in heifers, where often it is the only lesion found. Any of the glands may enlarge, but the favorite locations are the retropharyngeal, the submaxillary and others in the same region, the precrural, and the supramammary. An enlarged retropharyngeal gland may be palpated by extending the fingers forward above the larynx and bringing them together; or one may apply a speculum, insert the hand into the throat, and palpate the gland through the roof of the pharynx. On rectal examination enlarged glands may be found in the pelvic girdle and sublumbar region. A chronic painless swelling of any lymph gland should be accepted as positive evidence of tuberculosis until it proves to be otherwise. Tuberculous enlargement of the mediastinal lymph glands may cause tympany of the rumen from compression stenosis of the esophagus.

The Genitals.—In genital tuberculosis of the female the oviducts are often enlarged and indurated. Uterine tuberculosis may develop rapidly. In one of the author's cases a heifer freshened in March and had some bleeding from the uterus; in the following fall this organ was filled with quantities of gravel-like calcareous material. In diffuse tuberculous peritonitis, and in generalized forms, the oviducts are often involved. Vaginal exudate is positive to guinea pigs. Williams[15] states that he has not observed tuberculous orchitis or epididymitis, but has met with several cases of primary penial form. He regards infection of the vas deferens, seminal vesicle and prostate as possible, but apparently rare. Penial tuberculosis affects chiefly the submucosa of the glans, prepuce and sheath, and adjacent lymphatics. Every part of the genital organs of the cow is subject to invasion, though the ovarian form is rare.

Central Nervous System.—In the brain a tuberculous meningoencephalitis with caseation and sclerosis may form on the surface of the cerebri. The symptoms are progressive emaciation and paresis, hyperesthesia and motor irritation—walking in circles. In one of our cases the cow presented maniacal symptoms, similar to those observed in rabies.

Digestive Symptoms.—Ulcerative lesions in the intestines cause diarrhea and emaciation. Chronic bloat that develops after feeding and lasts for a few hours is typical of tympany of the rumen caused by compression of the esophagus by an enlarged mediastinal lymph gland. The *liver* may show pain on percussion when invaded by numerous tuberculous abscesses. The *peritoneum* may be found roughened on rectal examination and one may also palpate enlarged mesenteric lymph glands. In one case of diffuse peritonitis, gradual loss in condition and pain on percussion over the abdomen led to a mistaken diagnosis of traumatic gastritis.

Course and Prognosis.—When the rate of tuberculosis becomes high, losses from deaths, mastitis, joint disease, sterility, poor nutrition, etc., are usually heavy. But occasionally the disease may be widely distributed in a stable without causing serious loss. In any community where the rate is as high as 15 per cent the total loss is heavy. In the area covered by our ambulatory clinic the rate was formerly about 15 per cent, and the clinic records of that period show many calls to treat clinical cases of tuberculosis. At the present time we hardly encounter a single case in a year; the perpetual tax imposed by the disease is far greater than the cost of eradication.

Spreaders.—It is an important feature of bovine tuberculosis that when lesions develop in organs having open communication with the body openings, an almost constant exit of bacilli is taking place, regard-

less of the physical condition of the cow or the extent of the lesions. In a series of 262 tuberculous cows examined by Udall and Birch[16] nearly 20 per cent were found to be carrying tubercle bacilli in the esophagus as revealed by the sputum cup. Of 262 cows examined, 8.7 per cent showed symptoms and positive sputum, while 10.3 per cent showed no symptoms and positive sputum—many physical cases had been removed. This shows the futility of trying to distinguish between "open" and "closed" cases, or attempting to reduce the spread of infection by the removal of clinical forms. So long as the channels of infection remain open, one spreader is enough to seed the surroundings.

Tuberculin Test.—*Tuberculin.*—Koch's O. T. (old tuberculin) is prepared by growing the tubercle bacillus on glycerinated bouillon until growth ceases. Tuberculin is also made from cultures grown on synthetic media. Filter through porcelain, sterilize by heat, and evaporate to 10 per cent of the original volume.

Koch introduced tuberculin for the treatment of human tuberculosis, and it is still used as a remedy for certain forms of this disease. In veterinary medicine it is used exclusively for diagnosis.

According to Armsby and Pearson,[17] "tuberculin was first used in this country on cattle by the Tuberculosis Commission of the Veterinary Department of the University of Pennsylvania, of which Professor Zuill was chairman, and their report on the agent was favorable."

If tuberculin is injected subcutaneously into an animal with a focus of tuberculosis, there is a febrile reaction, sometimes a local and a constitutional one. If this injection is made into the skin, intradermic, there is a local reaction only. The effect of tuberculin on an infected individual is generally regarded as an anaphylactic reaction; the tuberculous process has made the individual hypersensitive to the proteins in tuberculin.

The *subcutaneous test* was introduced into the United States shortly after the discovery of tuberculin by Koch in 1890. Until March 1, 1920, it was the only test officially recognized by the Bureau of Animal Industry: *Tuberculin Testing of Livestock, Cir. 249,* U. S. Dept. Agr., 1936.[18] At the present time it has been almost entirely supplanted by the intradermic test. It has several disadvantages: (1) Tuberculous animals fail to react. This may be caused by previous injections of tuberculin. When animals are tested every six months with a dosage of not more than 2 cc., a considerable percentage of infected cows fail to give a temperature reaction. Finally the Federal Bureau adopted a 4-cc. dose, and this was often increased to 10 and even 20 cc. Cattle offered for sale were sometimes injected maliciously to prevent a reaction. When the subcutaneous test was in general use cows were given this

test for interstate shipment and then retested in 60 to 90 days in the same manner. This misuse of the subcutaneous method, in conjunction with its natural limitations, resulted in many misunderstandings and disappointments. In the experimental work done by Udall and Birch,[16] 55 individual subcutaneous tests of animals known to be tuberculous gave a failure to react in 27.7 per cent; these failures occurred irrespective of age or stage of development of the disease. The animals were tested every six months with a 2-cc. dosage. Before other methods were used, a 10 per cent reaction at each six-month or yearly test was common in herds, and complete failure was not rare. An old nonreacting spreader, often in excellent physical condition, would soon contaminate new recruits when mangers and troughs were exposed to her salivary droppings. (2) Healthy cows often carry high temperatures, and when these coincide with postinjection observations false condemnations occur.

Before starting a subcutaneous test, have a clear understanding with the owner in regard to the feeding, stabling, and number to be tested. Use not less than twelve high grade thermometers when testing alone. The thermometers should be 5 inches long, best quality, with a ring in the end for a 6-inch string, ¾ inch curtain ring, and 3½ inch rubber band ⅛ inch wide. Have a surplus of thermometers, syringes, and test charts. All animals should be restrained and in accustomed surroundings; provide an abundance of water; and feed roughage sparingly. Do not expect accurate results in animals that have received large doses of tuberculin within the past one or two years. Take at least three pre-injection temperatures, and omit from the test animals that are sick or show a temperature of 103°F. or higher. The dosage, according to regulations, varies from 2 cc. to 4 cc. for cows. Begin taking post-injection temperatures not later than the 8th hour and continue until the 18th hour after injection. When the highest recorded temperature of an individual occurs at the 18th hour, continue every two hours until the temperature falls.

According to the specifications of the Federal Bureau of Animal Industry, "a rise of 2°F. or more above the maximum temperature observed prior to the injection of the tuberculin or a temperature above 103.8°F. should be regarded as an indication of tuberculosis provided the temperature reaction shows the characteristic 'rainbow' curve. An elevation of temperature higher than 103.8°F. should be also regarded as an indication of tuberculosis even though the so-called rainbow curve is replaced by what is termed 'the plateau.'"

A sustained rise of two degrees, or above 103.8°F., is an indication of tuberculosis.

The Intradermic Test.—In 1907 Pirquet demonstrated that the injection of small amounts of tuberculin beneath the superficial layers of the skin caused a local redness and swelling at the seat of injection. The intradermic method has become the official test in the United States and has made possible the testing of large numbers of animals at slight cost. The chief advantages are: (1) Simplicity of operation. It has made possible the testing of all the cattle in a county or state at relatively slight cost. (2) In heavily infected herds, where large doses of tuberculin may have been injected subcutaneously, the intradermic method will leave fewer tuberculous animals. Cows are less readily desensitized because the dosage is small. Malicious interference with the test is not easy.

No-Lesion Cases.—When testing has eliminated most of the tuberculous animals in an area, the intradermic test may cause tail swellings that lead to condemnation of cows free of the disease. In such areas the percentage of "no-lesion" cases is high, and when several are taken from a purebred herd the result is criticized. For this reason one needs to be more conservative in the interpretation of the test in such herds than in those known to be highly tuberculous. While the saying, "Once a reactor always tuberculous," is true, it does not follow that all "reactions" are genuine. Hastings[13] has reported that in Wisconsin on a test of 30,010 cattle with 1.12 per cent reactions there were 22.5 per cent no-lesion cases. It has been proved that there are saprophytic organisms related to the tubercle bacillus which may invade the tissues and sensitize the animal to tuberculin, and that a part of the no-lesion cases are due to occult tuberculosis—either the lesions are incipient or they occur in a part of the body not usually affected or they may not be characteristic macroscopically. A certain percentage result from superficial or hasty application or observation of the test, or an effort to "read close" in order to detect every infected individual. In herds where the infection is slight or absent "close reading" may be disastrous.

When a herd has been tested repeatedly by the intradermic method and no reactors or visible lesions have been found, the appearance of a reactor on the intradermic test should lead to consideration of all the evidence available before the animal is destroyed; the most useful additional evidence is the subcutaneous test. Diseased cows that give a suspicious intradermic or eye reaction may react violently to a subcutaneous check test. When several animals in an accredited herd are suspicious on the intradermic test, and none shows a rise of temperature on the subcutaneous test, the result is negative. It is probable that the majority of no-lesion cases from accredited herds would not react to a subcutaneous check test. Unless one is willing to accept a high per

cent of no-lesion cases in accredited herds, all of the evidence available should be considered before condemnation. Hastings[13] has reported that 1,063 condemned cows from herds with only one reactor gave 44 per cent no-lesion cases. Bruner[19] has shown that as the percentage of reacting animals decreases, the percentage of no-lesion cases increases.

Mohler[12] reports "that the majority of animals which reacted to tuberculin and in which no lesions of tuberculosis could be observed on autopsy are from herds in which tuberculosis is present. It therefore seems probable that most of such animals are affected with bovine tuberculosis though the disease has not progressed sufficiently in them to be macroscopically visible. There are instances, however, of reactors to tuberculin being found in herds in which there is no history of tuberculosis. An excellent opportunity was presented to study reactors of this kind during the past year. A shipment of cattle from the island of Guernsey, supposedly free from tuberculosis, when they were retested at the port of entry at Baltimore, Maryland, had seven reactors." Three were posted and no lesions were found; an avian type of tubercle bacillus was recovered from the spleen of an inoculated guinea pig. In this report Mohler mentions that two of six cattle reacted after being fed human tuberculous sputum. Hastings and co-workers[14] conclude from their studies "that the observations made indicate that a positive response to tuberculin is not absolute proof of infection with tubercle bacilli. Some other of the mycobacteria may invade the tissue and sensitize to tuberculin."

Directions for Applying the Intradermic Test.—Materials required: cotton and alcohol for cleaning the tail fold; 35-minim syringe with a 1/4 inch 25-gauge screw needle. Wipe the tail fold clean with cotton moistened in alcohol. Inject not more than 1 minim of tuberculin into the skin near the ventral border of the tail fold about one-half the distance from its base. The New York State B. A. I.[20] recommends that 50 to 60 injections be made from 2 cc. (30 minims). Insist that the animal be properly restrained.

Reading the Intradermic Test.—At the end of about 72 hours observe the results and classify according to the following code: "—" negative; "X" slight reactions, 3/16 to 3/8 inch; "XX" marked reactions, 9/16 to 15/16 inch; extensive reactions, 1 1/8 to 1 7/8 inches.

The Double Intradermic Tuberculin Test.—For a number of years after the introduction of the intradermic method, it was applied in combination with the ophthalmic test. More recently it is used alone, or in combination with intradermic injection of the vulva. This is done by grasping the left vulva, at the lower portion, between the thumb

and finger of the left hand and injecting the tuberculin on the line of demarcation between the skin and mucous membrane half-way between the upper and lower commissures.

In so-called problem herds, where infected animals have not reacted to the usual tests, tuberculin is injected into areas of "marked sensitivity," such as the region of the neck, the cervical test. Swindle et al.[25] write that "the *cervical test* is of particular value in detecting tuberculosis in known infected herds and its use should be limited to such herds."

The Ophthalmic Tuberculin Test.—The ophthalmo-reaction of Calmette was introduced in 1907. It is made by placing a drop of concentrated tuberculin, or a disk of tuberculin in the eye. At the end of about two hours a second instillation is made, and six hours later the result is observed. A reaction is indicated by redness of the conjunctiva, swelling of the lids, and a free discharge of pus from the inner canthus. In large stables in the winter it is common to observe several individuals both before and after the instillation with a slight greyish or yellowish mucous discharge from the eye. As a result of the instillation a cow may develop a greyish discharge after four to six hours that somewhat resembles a reaction, but is more transient, disappearing after about two hours. A typical ophthalmic reaction with persistent conjunctivitis and purulent discharge is conclusive evidence of tuberculosis. The ophthalmic test has some value in special cases, but its routine use has been largely discontinued.

Examination of Excretions and Secretions.—As a supplement to the tuberculin test, guinea-pig inoculation of mucus taken from the esophagus with a sputum cup, has sometimes revealed open tuberculosis in animals that failed to react. Indication for the use of this method arises when, after repeated testing, the percentage of reactors remains high. Desensitization to tuberculin was not infrequent when subcutaneous testing was in general use. I have repeatedly found one or more open cases in herds directly after the removal of all animals reacting to the subcutaneous test. A report on this subject has been made by Udall and Birch.[16] Guinea-pig inoculations may also be made from vaginal excretions, feces, milk, and exudate taken directly from the trachea. Such examinations are applied for the detection of open cases of tuberculosis as a part of the Ostertag plan.

Methods of Control.—Since the discovery of the tubercle bacillus in 1882, and of tuberculin in 1890, various methods of control have been adopted in countries having an important dairy industry. In the United States tuberculosis was eradicated from many individual herds before the adoption of a country-wide campaign in 1917. But this had little effect on the general distribution of the disease. Exchange of

cattle, and change of ownership of farms, often returned the infection to its original status. Since the adoption of the *Accredited Herd Plan* supported by State and Federal appropriations, area testing, prompt disposal of reactors, adequate disinfection, and regulation of movement of cattle, have greatly reduced the areas of infection. According to the Report of the Committee on Tuberculosis of the U. S. Live Stock Sanitary Association, December 1938, "all counties in all states will have been declared modified accredited areas by December, 1939"; tuberculosis in a modified accredited area does not exceed one-half of 1 per cent. This is the most extensive animal-disease control program ever attempted.

In many instances failure to control the disease has resulted from lack of consideration of channels through which the bacilli flow. This has led to the construction of individual mangers and the use of individual drinking cups in stables where complete elimination of tuberculous cows proved to be unusually difficult. By this means it is possible to keep the disease within reasonable limits and to prevent widespread contamination before the nonreacting open cases are finally detected.

In Europe the *Bang Method* of control is reported as successful in the Scandinavian countries. Under this plan a herd is tuberculin-tested annually and all animals showing clinical tuberculosis are slaughtered. Nonclinical reactors are segregated. Calves are fed on sterilized milk or milk from nonreacting cows. Since reacting nonclinical cases of tuberculosis are often spreaders, the success of this plan depends largely on the intelligence and knowledge of the personnel, as well as the degree of separation of the reacting and nonreacting groups on the same farm.

The *Ostertag Method* depends on the earliest possible removal of cases of open tuberculosis without the aid of tuberculin. Since it is impossible to differentiate between open and closed cases with a reasonable degree of accuracy this method is of little value. From a standpoint of control, every cow known to be infected, though perhaps not diseased in the ordinary clinical sense, should be regarded as an open case.

"It is the *sources* of the tubercle bacillus and the *channels* through which these bacilli flow and are passed from one host to another that are or should be of chief importance and concern; for the prevention of tuberculosis is neither more nor less than preventing the passage of living tubercle bacilli from a tuberculous to a non-tuberculous animal or person" (Watson).[21]

Immunization Against Tuberculosis.—Koch found that when a guinea pig, with a subcutaneous focus caused by subcutaneous inocula-

tion, received a second injection, the primary focus healed and the pig survived. These observations brought hope that a cure for tuberculosis had been found, and for certain of the human forms this expectation has apparently been realized. Since 1890 one form or another of protective vaccination of cattle has been almost constantly advocated, but none has stood the test of time. Among these are various modifications of virulent and avirulent human tubercle bacilli (Behring, Klimmer, Koch); products of metabolism of tubercle bacilli (Heyman); and tubercle bacilli attentuated by growth of bile-containing media (Calmette and Guerin). At the present time the Calmette method for both man and cattle has many supporters in Europe. Experiments with the Calmette Method (B. C. G.) have been reported by Cotton and Crawford;[22] Watson;[23] and Haring, Traum, Hayes, and Henry.[24] Their results do not justify the use of this vaccine in America. Watson concludes that "a comparative study over a period of five or six years does not substantiate the claims made for B. C. G. vaccine as an efficient means of protection against bovine tuberculosis. . . . As the animals grow older and advance towards sexual maturity, the percentage in which tuberculous processes are developing increases, and animal revaccination, in many cases, has not prevented progressive disease."

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JOHNE'S DISEASE

(Chronic Specific Enteritis of Cattle; Paratuberculosis)

Definition.—Johne's disease is a highly fatal chronic infectious disease of cattle in which recurrent diarrhea and emaciation persist for months. On postmortem examination it presents typical thickening and corrugation of the intestinal mucosa. It is caused by a specific acid-fast rod which is similar in appearance to the tubercle bacillus. It is of chief importance in cattle, and may occur in sheep (Eveleth,[1] Taylor).[2]

History.—Apparently the disease was recognized in the eighteenth century, but it was not until early in the twentieth that it received careful study. In 1895 Johne and Frothingham[3] reported the finding of acid-fast organisms in intestinal lesions; they considered the disease a form of tuberculosis, and the acid-fast rods as avian tubercle bacilli. In Denmark a chronic incurable enteritis in cattle had been recognized for years previous to 1906, when Bang[4] announced that the acid-fast rod found in the lesions was neither an avian tubercle bacillus nor a degenerated form of the mammalian tubercle bacillus, but a different,

specific microorganism. As a name for the disease, which had previously been considered a special form of tuberculosis, he suggested *enteritis chronica bovis pseudotuberculosis*, or *paratuberculosis*. He transmitted the disease experimentally by feeding diseased intestinal mucosa. His description of the morbid anatomy and symptoms is remarkably complete. He speaks of the long period of incubation, the slow spread, the recurrent symptoms, the occasional rapid gain in flesh, the possible recovery from light attacks, and the incurable nature of severe forms. Subsequent observers have added little to his description of the symptoms and lesions. In 1907 M'Fadyean[5] reported upon the occurrence of the disease in England; he confirmed the work of Bang with respect to the specific etiology and nature, and gave to it the name Johne's disease. The bacillus was first grown in pure culture by Twort,[6] in 1911. The disease was first described in the United States by Leonard Pearson.[7]

Etiology.—Johne's disease is widely prevalent in Europe and England, including the Channel Islands. Beach and coworkers,[8] give an estimate of 1 per cent infection in England, and report its occurrence in America in 27 states, including 8 herds belonging to Colleges of Agriculture, and 76 Wisconsin herds. Lash and Mohler[9] wrote in 1930, "There is no doubt that the infection has spread to virtually every State," and Johnson[10] added in 1945 that the disease which is already widespread, is increasing. In its distribution, the disease is chiefly a serious endemic in certain herds; it is not thickly scattered through the herds of any given area in this country.

In 1949 the principal centers in the United States were reported to be: New Jersey, Ohio, Indiana, Wisconsin, Washington and Oregon, central Minnesota, and the northwest corner of California. South Dakota, Missouri, Kansas, Oklahoma, Utah, Idaho, Arizona, and Wyoming are exempt. Elsewhere the infection is sparsely scattered.[24]

Because of the presence of affected, as well as infected animals which are permanently non-clinical, the estimated prevalence is probably too low.

On *acid soils* the symptoms of the disease are more active than in areas where the soil is alkaline.

A *seasonal* increase in summer is mentioned by Hess (Bern), who states that it is chiefly observed in cattle at pasture. Because of an apparent increase during the summer which he revealed by tests conducted in the spring and fall, Hagan[11] has suggested the possibility of reinfection from the pasture.

The *age* when symptoms are most often observed is from two to six years. Infection readily occurs in younger animals, but on account of

the long period of incubation, from six months to a year, physical signs are rarely met with until the animal approaches maturity. Calves often react to johnin. In a test conducted by Ernest,[12] "a surprising number of young animals reacted. In one lot of twenty-one calves, ranging in age from three weeks to six months, there were found seventeen reactors and two positive suspects." In conducting transmission experiments, Meyer[13] was more successful when young animals were used, and he concluded that "the natural infection probably takes place in the early days of life by contact with infected mothers or surroundings. The possibility of infection in certain pastures, where *M. paratuberculosis* leads a saprophytic life, cannot be denied, and it is probably of importance for the infection in adults." In the monograph by Twort and Ingram[14] it is reported from the Channel Islands that mature healthy cows (5 to 6 years old) when put on infected farms do not develop the disease, and that the malady is chiefly confined to low-lying districts. In a herd in New York, where there had been five deaths from Johne's disease in the previous five years, Udall tested with johnin intravenously the entire herd of 20 adults and 8 calves. Nine reacted, and six of these were 2-year-olds; the other ages were 3, 4, and 7. Nearly all of the reactors presented physical symptoms. There is an age immunity.

Bacteriology.—*Mycobacterium paratuberculosis* (Johne's bacillus) so closely resembles the tubercle bacillus that positive differentiation under the microscope is difficult, but it may be indentified by its smaller size and its arrangement in clumps in the tissues and feces. The acid-fast and alcohol-fast characteristics are identical with those of the tubercle bacillus. It is difficult to culture, and aberrant strains are frequently observed. Hastings, et al.[8] state that the frequency of isolation of what are apparently avian tubercle bacilli from supposed lesions of Johne's disease deserves consideration, and he finds further suggestion of a relationship in the fact that avian tuberculin is used as a diagnostic agent for Johne's disease.

Its *habitat within the animal* is in the diseased mucosa and the mesenteric lymph glands. In histological sections of affected tissues it is present in great numbers. Detached particles of the mucous membrane of the rectum may show many of the bacilli in stained smears. Animals may harbor the infection for years and yet show no signs of the disease. In culturing the glands of cattle, rabbits, and swine, Hastings et al.[8] observed that one may pick up acid-fasts almost anywhere in glands that show no microscopic evidence of the disease, and from animals that apparently are in perfect health.

The bacilli are eliminated in the feces, and, so far as is known, in

no other manner. It is probable that they are being eliminated long before there are any symptoms. It is estimated that 40 to 50 per cent of the affected animals are giving off the organisms. The *habitat outside the body* is unknown. In experiments conducted by Lovell, Levi, and Francis[21] on the survival of Johne's bacillus, naturally infected feces exposed to atmospheric conditions contained viable bacilli at 246 days but not at 284 days. According to Bang,[4] the disease sometimes dis-

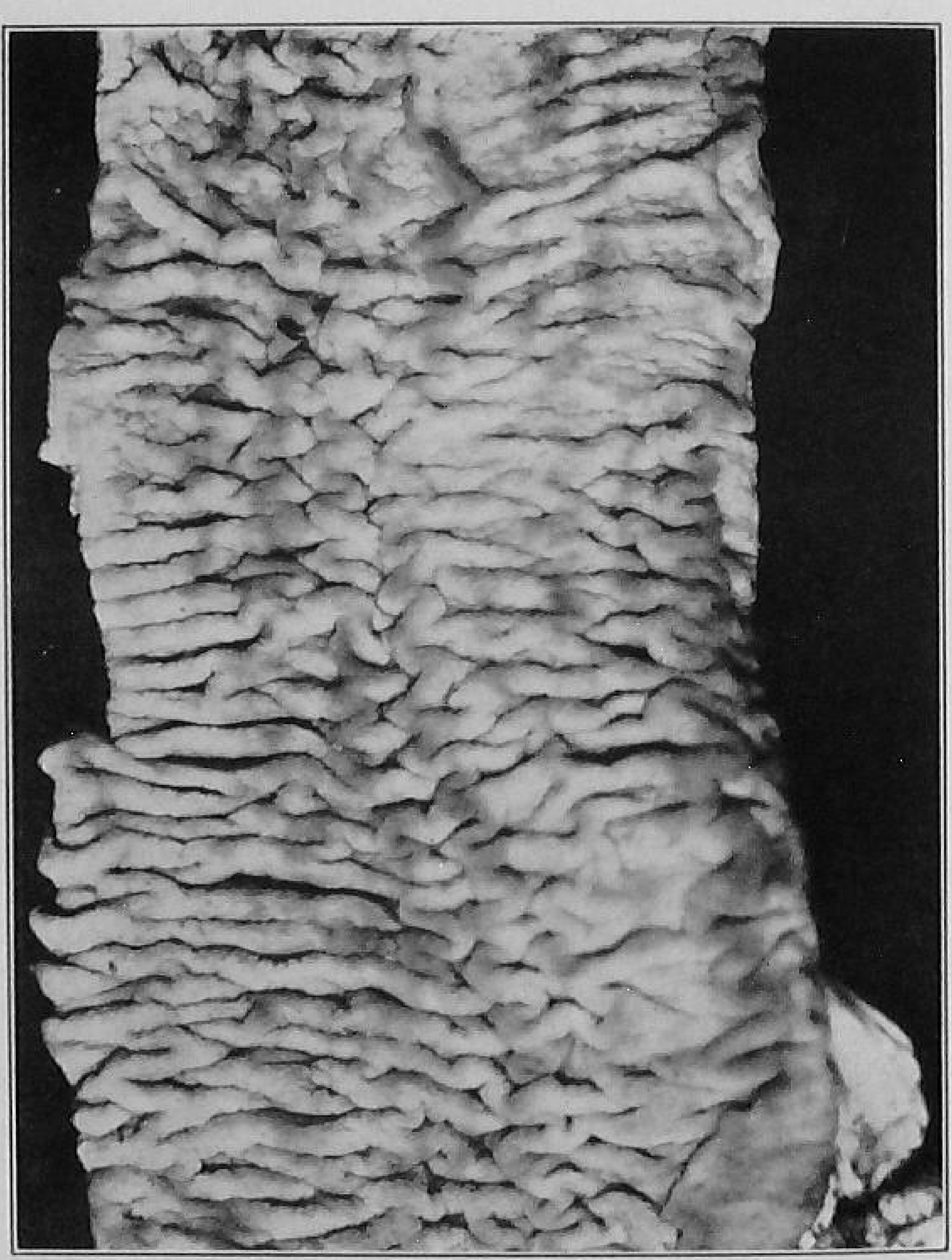

Fig. 89.—Mucous membrane of the ileum in Johne's disease. (Hagan and Zeissig, Rep. New York State Veterinary College, 1927-28.)

appears spontaneously. On the other hand, the spread may be rapid.

Artificial inoculation is not generally successful in any of the laboratory animals, though infection may occur when massive doses are given. Hagan and Mansfield[15] were able to induce peritoneal lesions of the great omentum regularly in guinea pigs by means of intraperitoneal injection of the bacillus. The disease may be caused in cattle in its

typical form by intravenous injections or by the feeding of culture or lesions. Natural infection undoubtedly takes place through the digestive tract. And while the bacillus has never been recovered from sources outside the body, the wide distribution of the disease under so many different conditions of housing and care leaves no doubt that it survives wherever fecal contamination occurs.

Morbid Anatomy.—The cadaver shows extreme emanciation, the fatty tissue being greatly reduced and somewhat gelatinous. On opening the abdominal cavity the small intestine appears to be thickened even from external observation; this thickening is in sections separated by normal parts, and it is most marked in the terminal portion. On

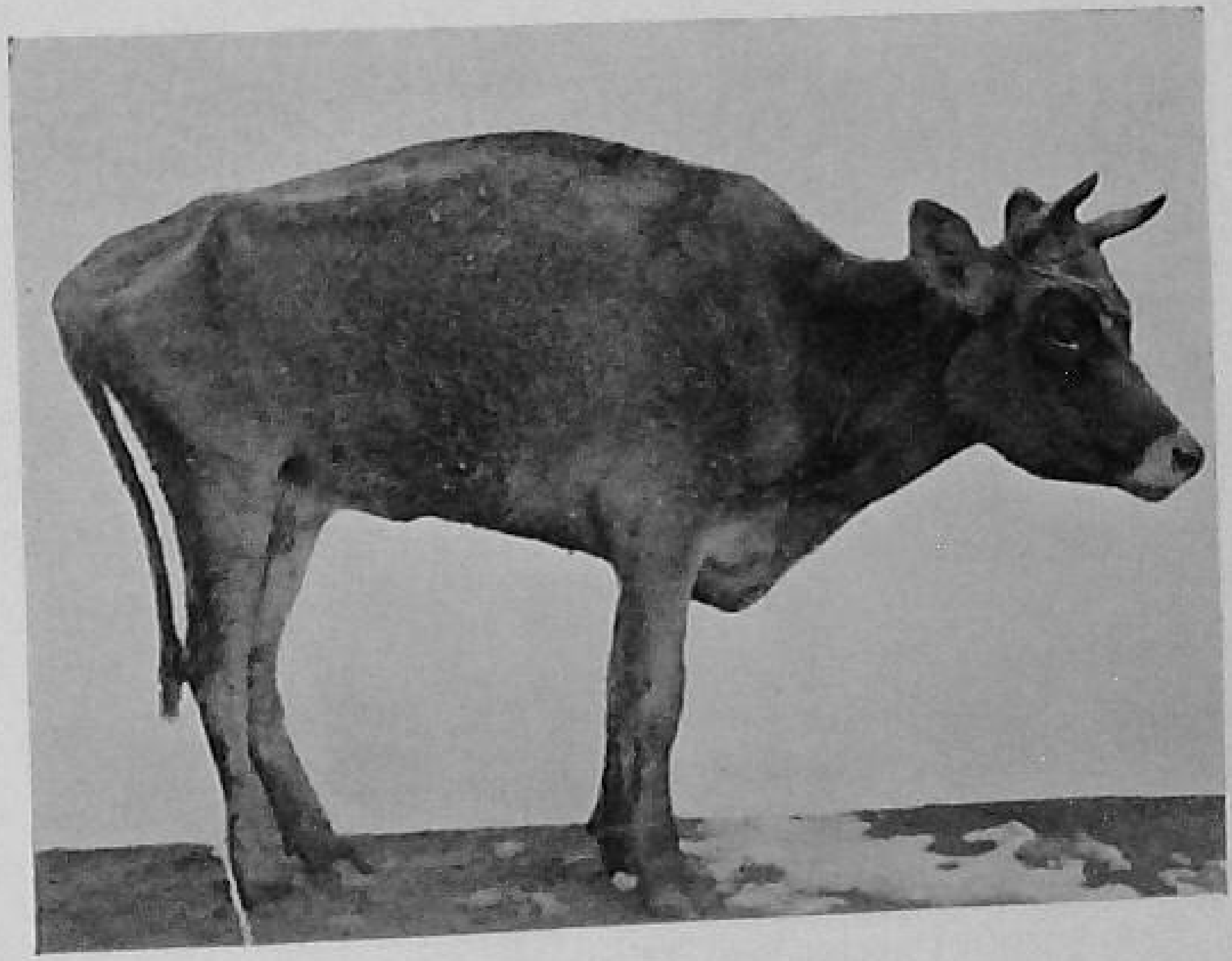

Fig. 90.—Johne's disease.

opening the affected parts of the small intestine, the mucosa is found to be thickened three to four times its normal thickness and thrown up in irregular folds. On the surface of the folds, the mucosa is smooth, but in the depths it is somewhat roughened and eroded, though extensive loss of substance does not occur. In rare instances involvement of the entire intestinal tract may be met with, yet in advanced clinical cases the changes in the intestinal wall may be so slight as to pass undetected unless one is looking for Johne's disease. The cecum is frequently affected in the form of patchy corrugations. In the writings by Beach and Hastings[8] they emphasize the frequency of inflammation of the ileocecal valve, stating that it may be greatly swollen, fifteen to twenty times its normal size; in several reacting animals, not showing symptoms, the ileocecal valve was markedly enlarged and inflamed. The mesenteric lymph glands are usually enlarged and moist, but otherwise

without macroscopic changes. In the absence of macroscopic evidence of the disease, acid-fast bacilli may be demonstrated microscopically. But in view of recent observations on the saprophytic occurrence of acid-fasts in glands and animals apparently normal, a diagnosis of disease on their presence alone seems hardly justified.

Microscopic changes.—The intestinal villi show marked changes; they contain nests of large epithelioid cells containing single or multiple nuclei. Around the cell-nests is a zone of leucocytes and lymphocytes. In the lymph glands are found identical epithelioid or giant cells. Sections of the intestinal wall or of a lymph gland stained according to the Ziehl-Nielson method usually show many acid-fast organisms arranged in groups. Necrosis and caseation do not appear. The change is identical with that of the initial stage of a tubercle, and like the tubercle, "presents in its early formation nothing distinctive or peculiar, either in its components or in their arrangement."—Osler.[16]

Symptoms.—The period of incubation is estimated at from six months to a year, and in many instances it is considerably longer. The frequency of Johne's disease at the age of two and three years is strongly suggestive of infection as a calf. Infection seems to occur much less readily than in bovine tuberculosis; usually not more than one or two cases occur on a farm in a year. Beach[8] cites an instance where three animals were introduced into a herd in 1910; two were sold in 1912 because of symptoms; the third was sold in 1913 for the same reason. In the period 1913 to 1921 fifteen animals were removed. In another herd it was two years after introduction of an infected animal before others showed physical signs. The first distinctive symptom is chronic, fetid diarrhea without straining. The appetite is usually good, and the attitude lively. The milk flow is decreased. The temperature is normal, and the condition gradually becomes poor. The pulse is said to be somewhat stronger than normal. If the diarrhea is checked by means of medication or dry feed, it soon returns. Without any treatment the diarrhea may stop for months, the feces being normal. In such cases the nutrition improves. Bang[4] noted a gain of 50 pounds in a month in one case. Peculiar nervous symptoms are mentioned by both Bang and Hess. The diarrhea is aggravated by a diet of green food, such as beets or pasture. Finally emaciation becomes extreme, the eyes are sunken, and recumbency is constant.

Often the disease becomes active after parturition, a suggestion that it has been latent for months, possibly since exposure as a calf. It is doubtful that an infection leading to clinical symptoms is ever overcome. Deaths from Johne's disease have been observed in cows whose feces remained normal through the entire course.

Diagnosis.—Chronic recurrent diarrhea associated with a poor general condition is strongly suggestive of paratuberculosis. In herds where the infection is known to exist, the appearance of this syndrome following parturition is almost conclusive. On rectal examination one may encounter the typical corrugations on the mucosa, but this is unusual. Microscopic examination of a piece of mucosa removed from the rectum with the finger nail may reveal groups of acid-fast bacilli, and "an experienced observer usually can be sure of the identification of the organisms seen."[23] If there is no other evidence of the disease, the significance of acid-fast rods is uncertain. Negative results do not exclude the possibility of infection.

Johnin (paratuberculin) was first prepared by Twort and Ingram[6] shortly after they succeeded in growing the bacillus in pure culture. It is made from Johne's bacillus after the method used in the manufacture of tuberculin. Because of the difficulty of obtaining abundant growths on media its preparation is expensive. According to Ernest[12] johnin gives temperature reactions that are about what one would expect in tuberculosis from tuberculin. Beach[8] has used johnin extensively in Wisconsin and reports it to be more successful than avian tuberculin. It is claimed of johnin that it does not cause reactions in cattle affected with tuberculosis. In 1945 Johnson[10] reported that intradermal johnin with a potency equal to that of mammalian tuberculin is now being prepared, and that in many instances the johnin used in the past was not highly potent. But Hagan and Zeissig[17] maintain that johnin will cause reactions in cattle sensitized to avian tubercle bacillus, and Hagan[11] has found all johnin products inferior to avian tuberculin administered intravenously. While johnin reveals many infected animals its use has not led to eradication of the disease from infected herds. Whether this is due to failure by johnin to reveal infected animals, or failure by man to control the sources of infection, is yet to be demonstrated. The technic of the intravenous johnin test is as follows:

Take three preinjection temperatures at two-hour intervals. Omit animals with a temperature of 103°F. or higher. Inject the prescribed dose intravenously immediately after the third preinjection reading. Resume the temperature readings one hour after the injection and continue every hour for twelve hours. A rise in temperature may occur as early as one hour after injection, but the majority of reactions are observed in from three to eight hours. A rise of 1.5° or more above the preinjection temperature constitutes a reaction, but this elevation should continue for at least three hours, and it may persist for twelve hours or more. In general the rise in temperature is less marked than in the tuberculin test for tuberculosis, but the physical reaction is

greater. The majority of reactors show a rough hair coat in from one to four hours after receiving johnin. At the height of the reaction scouring is frequent; according to Beach[8] this is present in about 25 per cent of the affected cows. Sometimes chills and dyspnea appear in from fifteen to thirty minutes after the injection and persist for from one to two hours.

Relations of Reactions to Morbid Anatomy.—Hastings, Beach, and Mansfield[8] have reported the autopsies of 24 reactors to the johnin test. The results were as follows:

Normal ileocecal valve, intestinal lesions marked	2
Marked involvement of the ileocecal valve and intestine	7
Slight involvement of ileocecal valve and intestine	4
Marked involvement of the ileocecal valve only	4
Slight involvement of the ileocecal valve only	3
No changes	4

Thus there were no macroscopic changes in the intestine in 45.7 per cent. On the other hand, Beach reports finding acid-fast organisms in 36 of the 37 reacting animals examined.

The limitations of johnin as a diagnostic agent are shown by the New Zealand Report[18] for 1938 in the statement that "this chronic bacterial infection of cattle presents very great difficulties in the matter of control, a most disappointing feature being the occurrence of further cases on certain farms on which the entire herds have been subjected to twice-yearly tests with the diagnostic agent, johnin. It is therefore apparent that, on at least a percentage of farms, the eradication of Johne's disease presents most formidable difficulties."

Avian Tuberculin.—Because of the similarity of the avian tubercle bacillus to Johne's bacillus, avian tuberculin was used by Olaf Bang[19] as a diagnostic test in 1908. The results led him to believe that a diagnostic method, comparable to that of the test for tuberculosis, had been found. In 1910 he reported its use on 1,700 cattle with a failure of 7 per cent as revealed by 50 autopsies. But there is little to indicate that avian tuberculin has contributed materially to the control of Johne's disease since it was first used as a diagnostic agent about twenty-five years ago. Hagan and Zeissig[17] have reported on the relative reactions of avian tuberculin and johnin when injected by various methods into different species of animals. They concluded "that when applied to cattle and other animals naturally or artificially infected with the organisms of Johne's disease, avian tuberculin apparently has given results identical with those obtained with johnin." In their hands both avian tuberculin and johnin have failed more often than they have

succeeded in eliciting reactions in animals in advanced stages of the disease.

Methods of Control.—While johnin and avian tuberculin have been in use for years, there are comparatively few autopsy reports to indicate their degree of accuracy as diagnostic agents under field conditions. Furthermore, little is known of the extent to which reactions fail to occur in infected cattle. It has been predicted that the disease will finally reach the same degree of concentration in the United States that it has attained in certain parts of Europe. Under the provision of the Federal Government that indemnity will be paid for reactors, there have been few claims, and there is little to indicate whether the disease is more or less prevalent now than it was twenty-five years ago. It is probable that improvement in sanitation attending the control of bovine tuberculosis has helped to check the interherd distribution of Johne's bacillus.

There is every indication that the calf is the chief soil for new infection and that the new-seeding is largely in the first year of life. Thus the combat against this disease adds another to the numerous reasons why the calf should be removed from its dam after birth, and permanently separated from adults until it reaches breeding maturity.

The paths of exit from the body are not nearly so numerous in the case of Johne's disease as they are in tuberculosis, since it escapes only in the feces. In its epidemiology, the dissemination is so slow and the disease so insidious that it may become established in a herd before its presence is revealed. Nonclinical carriers may be added by purchase. Thus the disease requires, even more than tuberculosis, the practice of stable and pasture hygiene directed against the spread of an unrecognized infection within the herd, a hygiene that will prevent the ingestion of bacilli which may reach the feces. Fundamentally, it is the prevention of fecal contamination of food and water. In the case of water this is readily accomplished in the important dairy states of the Middle West and East. Stagnant water in holes or streams needs to be avoided. Mangers and feed racks need to be so constructed that the animal does not eat off the ground, and mangers should be protected against sweepings from the stable floor. It is improbable that any diagnostic test will ever replace the effect of hygienic herd practice in the control of this disease.

The failure of the campaign against Johne's disease is shown in the following interesting quotation from Larson, Beach, and Wisnicky,[20] of Wisconsin, where efforts against the disease have been especially active:

"Field experience with the control and eradication of Johne' disease is not very encouraging. Even with the present diagnostic agent, johnin,

considered far better than the product which was originally made, it is extremely difficult to eradicate the disease from a herd. Perhaps the test itself is as efficient as one would expect a diagnostic test to be, but the fact remains that with our present test and with such knowledge as we have relative to the disease, and the procedure to be followed in eliminating it from a herd, we find it extremely difficult, frequently impossible, to rid herds of the disease. . . . In reviewing our work in a considerable number of herds, we can look back to only one heavily infected herd in which, at the present time, we have reasonable grounds to believe that the disease may have been eliminated. . . . It is not feasible to quarantine since the owner cannot be assured that the disease can be eliminated. . . . As it works out infected herds are not prevented from being disseminated. . . . It would be of interest to condemn entire herds. . . . In time it will be too late. . . . Cattle are consigned to sales from herds known to be infected. . . . The disease is never considered by purchasers of stock at sales. . . ."

A method of control suggested by the report of Lovell, Levi, and Francis,[21] that naturally infected feces experimentally exposed to atmospheric conditions did not contain viable bacilli at 284 days, led Marsh[22] to a trial with an infected ranch from which all cattle had been removed and which was not restocked for approximately one year; the results indicated that premises could be freed of the infection by this procedure. This was followed by an experiment with sheep upon which the following summary was reported: "The history of this outbreak of Johne's disease in sheep indicates that where sheep run as a farm flock become infected with Johne's disease, the infection can be eliminated from the farm by disposing of all the sheep, and leaving the area free from sheep or cattle for one year before restocking."[22]

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ACTINOMYCOSIS

(Lumpy Jaw; Wooden Tongue; Big Head)

Definition.—Actinomycosis is a chronic infectious disease affecting chiefly cattle and swine and caused by *Actinomyces bovis;* in cattle it attacks the bones of the head, causing rarefying ostitis; in swine it causes mastitis. Actinobacillosis is a chronic infectious disease affecting chiefly the soft parts of the head of cattle; it is characterized by abscesses and fistulae with thick connective-tissue walls and is caused by *Actinobacillus lignieresi.* Pus from affected tissues contains charac-

teristic yellowish granules (rosettes) from 0.5 to 1 mm. in diameter surrounded by radiating club-like terminations.

Etiology.—*General Prevalence.*—In the United States actinomycosis has been widely prevalent in the dry Western Plains of the Mississippi Valley and in the states west of the Rocky Mountains. As a sporadic and endemic disease it is frequent throughout this country. It is especially frequent on certain farms and in certain localities. In the ambulatory clinic of the New York State Veterinary College several cases are treated each year, and it is probable that in its general distribution the disease is equally prevalent in all parts of the United States. It is said

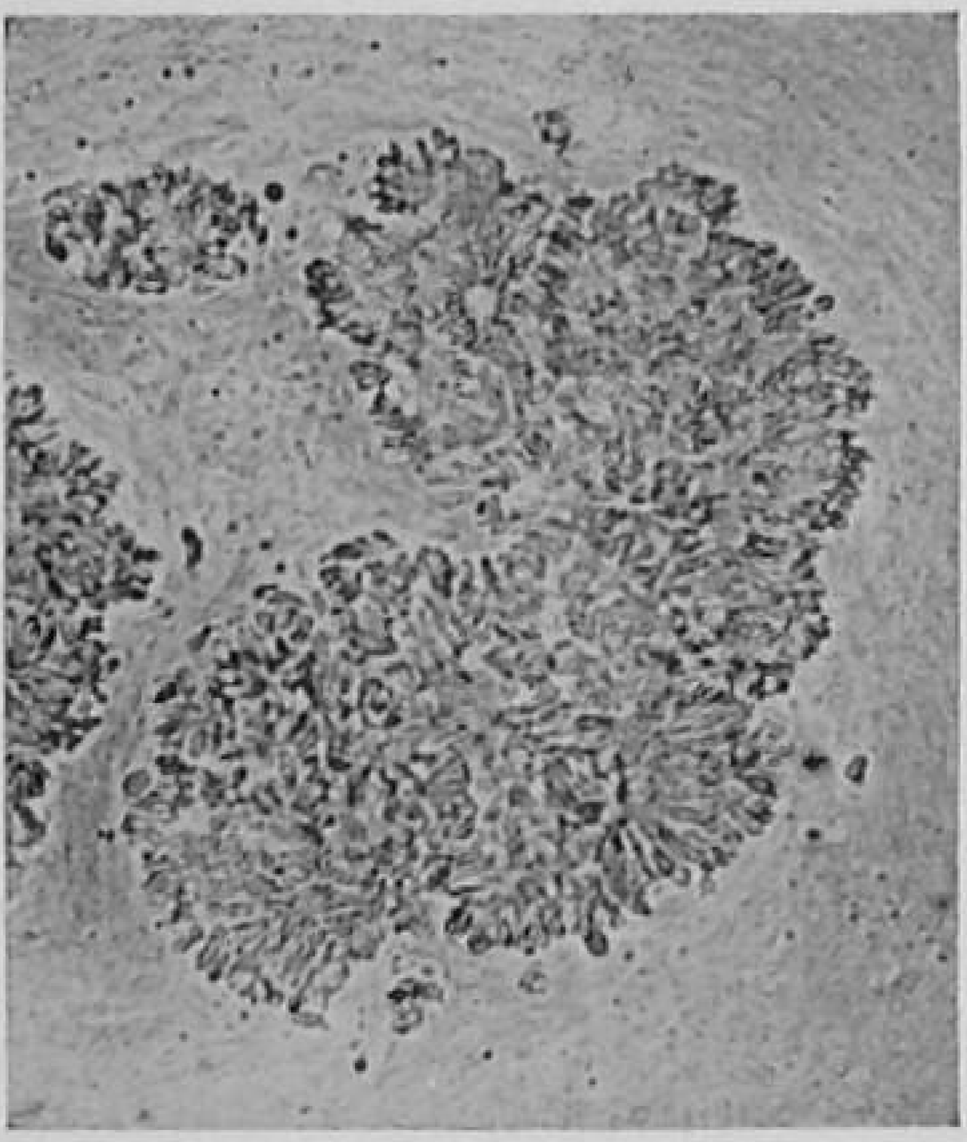

Fig. 91.—Actinobacillosis, club-bearing rosettes in pus from lymph gland. × 360. (Courtesy of L. R. Vawter.)[5]

to be most frequent in low-lying districts, but there is little evidence to support this statement. Such an influence could hardly be applied to the Western Plains where the pastures are both high and dry and where actinomycosis is said to be frequent. The statement that cattle fed upon coarse roughage are more subject to the disease may also be questioned. In its cyclic occurrence in certain sections and on certain farms, it resembles contagious diseases in general. Apparently it is more prevalent in the Southwest and West than in the East. Magnusson[1] estimates that at the slaughter house in Malmo 25 per cent of the older sows have actinomycosis of the mammary glands. The age incidence is chiefly from 2 to 5 years.

Bacteriology.—*Actinomyces bovis* (The Ray Fungus) was first described by Wolff and Israel[2] in 1891. In 1938 Emmons[3] reported the

finding of *A. bovis* in humans in 37 per cent of 200 pairs of tonsils from routine tonsillectomies. He writes, "It seems probable that *Actinomyces bovis* is commonly present in the normal mouth and throat, and becomes pathogenic only under extraordinary conditions. The tonsillar crypts serve as an important reservoir for the saprophytic phase of this pathogenic fungus." It is also probable that *Actinomyces bovis* is commonly present in the normal mouth and throat of animals, and that this is its chief habitat. In gross appearance actinomycotic pus is viscid, yellowish, and contains yellowish granules ("sulfur granules") which vary in size from minute particles to bodies 4 mm. in diameter

Fig. 92.—Branched filaments and coccoids in pus from bone actinomycosis, Gram stain. × 900. (Courtesy of L. R. Vawter.)[5]

that are visible without magnification. Pus from lesions of actinobacillosis is similar in appearance, but the granules are smaller and not usually visible without magnification. Actinomyces is described by Magnusson as a gram-positive filamented microorganism capable of forming true branchings and easily breaking up into short segments and granules. In describing the granules of *Actinomyces bovis*, Magnusson writes: "The most striking picture is seen in preparations stained according to Gram. We see large and small cocci, short and long, fine and coarse filaments. Small granules lying in the form of chains resembling streptococci, but also distinctly shaped short rods. Very often we see distinctly shaped branched filaments, so-called mycelium with true branching. But on closer examination we see that the numerous cocci and small granules are nothing but broken-up filaments."

Magnusson was successful in transmitting this infection by means of artificial inoculation of pus and cultures in about 25 per cent of trials. Infection is thought to enter the jaw through the alveoli of the teeth or an ulcer in the cavity of the mouth. This theory is supported by the occurrence of the disease chiefly in young cattle before the teeth become permanent. The infection does not spread through the lymphatic system and it is not known to attack the lymph glands.

Actinobacillus lignieresi (Lignières and Spitz)[4] is the usual cause of actinomycosis of the soft parts of the head, if the bone is not involved; these include the tongue (wooden tongue), lips, gums, palate

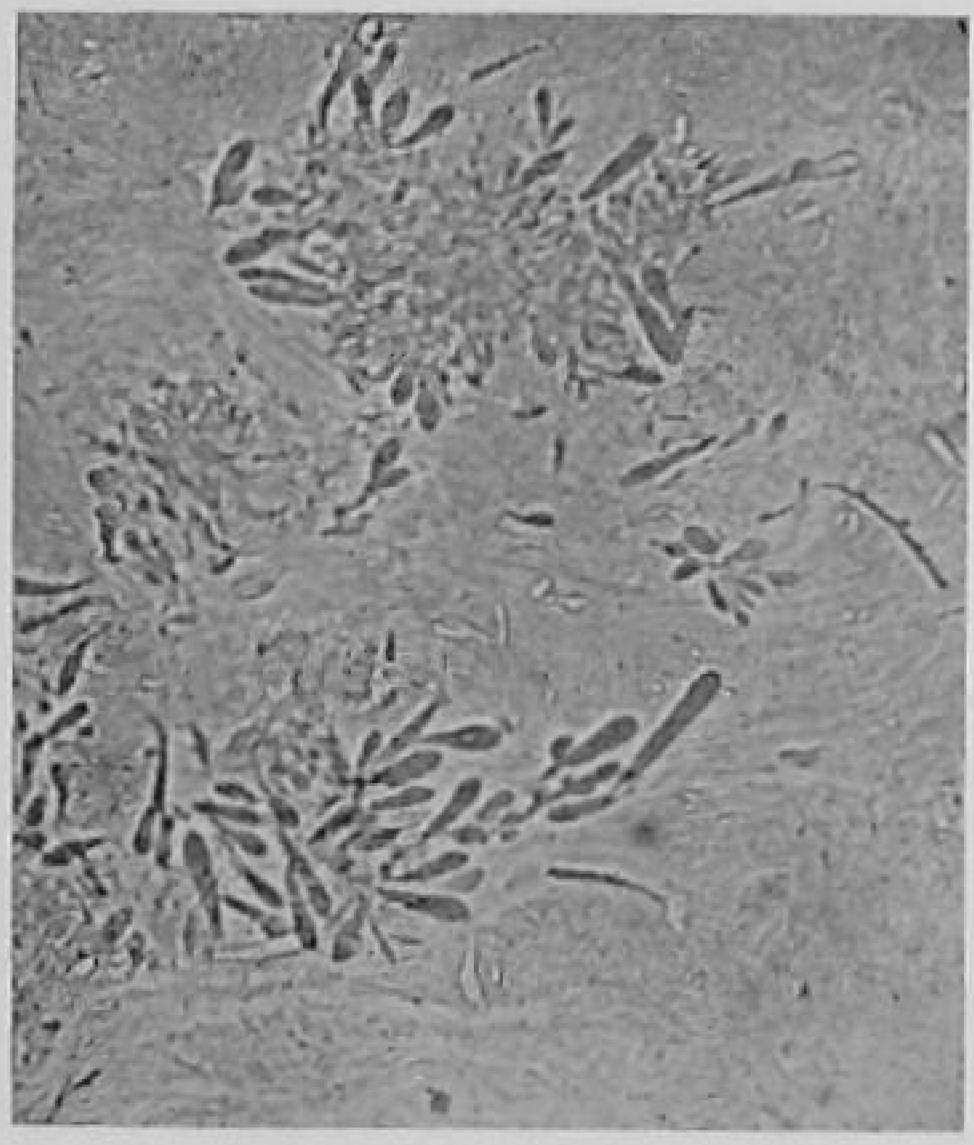

Fig. 93.—Clubs and rosettes in pus from actinomycosis of mandible. × 360. (Courtesy of L. R. Vawter.)[5]

and adjacent lymph glands. In addition it may cause lesions in the larynx, esophagus, rumen and reticulum, liver, lungs, and serous membranes. The mouth is thought to be its normal habitat. It is a gram-negative rod that may appear in long chains in the pus granules. In these granules there are no central radiating filaments; the center consists of clusters of gram-negative bacilli, and the radiating clubs are smaller and more numerous than in *Actinomyces bovis*. This disease is easily reproduced by means of artificial inoculation with pus or cultures, and natural infection enters through the mucous membrane.

Thus actinomycosis of the maxillary bones and most cases of actinomycosis of the udders of sows is genuine actinomycosis, while that affecting the soft parts of the head, the digestive and respiratory tracts of cattle is chiefly actinobacillosis.

Mode of Infection.—Ulcers on the oral mucosa and around the alveoli of the teeth are regarded as the port of entry of *Actinomyces bovis* into the maxillary bones. The theory is supported by knowledge of its habitat in the mouth and by the prevalence of the disease in young animals during the period of change of teeth. Recurrent endemics in groups of young cattle on the same farm at intervals of three to five years, are suggestive of contagion. Such a view is supported by the positive results of Magnusson's transmission experiments, and by the epizootic actinobacillosis described by Lignières and Spitz.[4] There are numerous instances, however, where the disease affects only one animal in the herd.

In actinomycosis of the soft parts of the head, such as the subcutis of the submaxillary and throat region, the tongue, and the pharyngeal ring, alveolar periostitis in association with shedding of teeth cannot be regarded as a predisposing cause. Yet the age incidence is the same, 2 to 5 years.

Infection of the mammary glands of swine has been attributed to the intimate contact of this organ with infected straw. Since it has been shown that the species of actinomyces infecting the mammary glands of the sow is not saprophytic on grain awns and straw, but is parasitic in the mouth cavity, the theory has been advanced that infection is caused by the sharp teeth of suckling pigs, a wound infection.

Pathological Changes.—There are two chief groups of anatomical changes in actinomycosis: those associated with clinical forms of the disease, and those found only on postmortem examination of apparently healthy slaughtered stock.

In cattle the most frequent seat of lesions is in the *bones of the jaw*. Where these lesions have led to death or disposal of the cow because of extensive growths, there is usually a marked bony swelling of both the lower and upper jaws. On the surface there may be numerous small fistulous openings that communicate with the underlying spongy bone. The surface may also present one or more granulomatous suppurative growths of soft tissue. In the mouth cavity the gums and palate are extensively swollen, the teeth are often loose, and the corresponding alveolar periostitis is accepted by most authorities as the origin of the infection. When the bony swelling is split with a saw, the interior is found to consist of spongy bone (rarefying ostitis, "osteosarcoma") infiltrated with pus. The lesions are purely local. When the lymph glands contain lesions, infection may be attributed to actinobacillus rather than actinomyces.

The lesions of clinical forms of actinobacillosis of the soft parts of the head and neck are chiefly of the nature of an infectious granuloma

containing foci of pus, or there may be typical abscess formations. These growths may be located anywhere in the *submaxillary region.*

A far less frequent, but more serious, location of lesions is within or outside the *pharyngeal ring*. Actinomycotic abscesses in the soft tissues outside the pharyngeal or laryngeal wall are small, multiple, and thick-walled. On the mucosa of the larynx and pharynx they are found as polypoid growths containing pus, or as granulations. *Actinomycosis of the tongue* occurs in the posterior part; it is enlarged and ulcerative on

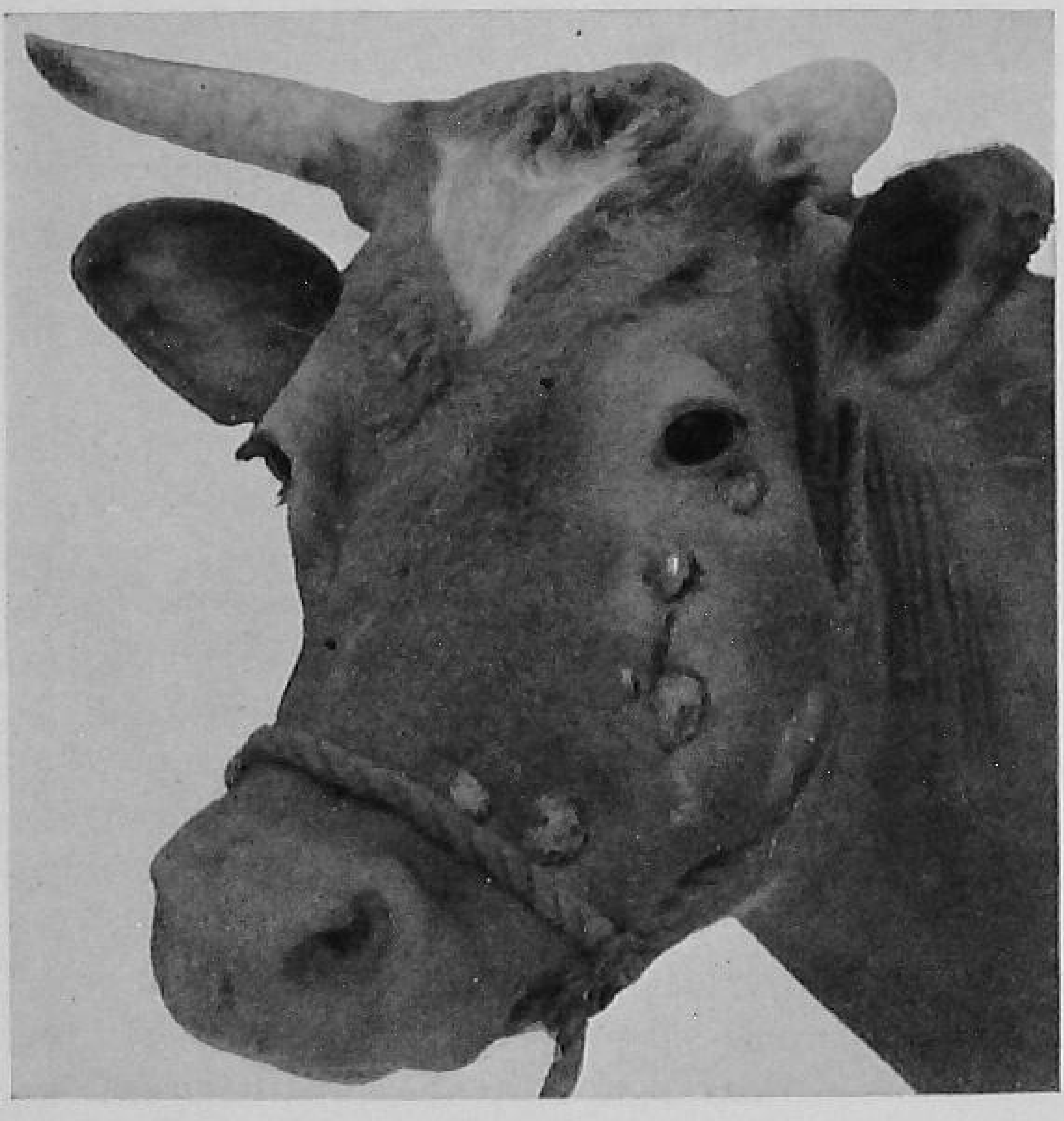

Fig. 94.—Actinomycosis.

the dorsal surface. Often there is marked thickening and induration of the muscular tissue (wooden-tongue). The submucous tissues of the lateral surface may contain numerous small actinomycotic nodules; there may also be larger and deeper abscesses. In association with lesions in the mouth and throat one often finds an involvement of the submaxillary and retropharyngeal lymph glands; they are swollen and contain actinomycotic foci made up of grayish-red soft walls and an interior of thick creamy pus. All of these changes in the soft tissues are attributed to actinobacillus. In the pus one finds numerous granules which present the typical rosette form with well-defined clubs, but the granules contain no gram-postive elements.

In the *udder of the sow* there are many small abscesses embedded in the dense fibrous connective tissue areas. Incision of the infected tissue discloses nodules from one-fourth to one inch in diameter; these contain pus filled with calcified granules. On the surface of the mammary glands are fistulous openings and granulating ulcers.

Internal actinomycosis is met with in slaughtered animals not showing clinical signs. The causative agent is *Actinobacillus*. The lesions are found in the rumen, reticulum, lungs, pleura, peritoneum, liver, and adjacent lymph glands. According to Davies and Torrance[6] differentiation of these lesions from tuberculosis may be impossible on macroscopic examination. In 1921 Beaver[7] reported that next to tuberculosis, actinomycosis was the most common specific pathological condition seen in cattle slaughtered at the abattoirs of South St. Paul, and that while it usually involved the head, it was not infrequent in the tongue or lungs; a case of actinomycosis of the omentum was described. Moore[8] has called attention to the error of confusing pulmonary actinomycosis with tuberculosis, and the fact that actinomycosis may attack any organ in the body.

Symptoms.—Actinomycosis of the maxillary bones comprises about two thirds of the cases met with in our ambulatory clinic, and at least three fourths are between the ages of 2 to 5 years, regardless of the location of the disease. According to Davies and Torrance[6] *Actinobacillus* is the cause of 70 to 80 per cent of clinical actinomycosis in cattle.

Actinomycosis of the bone first appears as a circumscribed immovable bony swelling on the upper or lower jaw; as a rule this swelling corresponds to the position of the third or fourth molars. In our series the left lower jaw has been the most frequent location. Often the owner thinks it has been caused by a blow. The actinomycotic growth may enlarge rapidly, involving a large portion of the face within one to two months. In such cases examination of the mouth reveals a swelling of the hard palate, and, in some, loosening of the molar teeth. Swelling of the nasal bones may cause inspiratory dyspnea. Eating is difficult. Rapid growth of the swelling soon leads to emaciation and slaughter. As a rule the rarefying ostitis develops more gradually; after six to eighteen months growth may entirely cease, leaving a swelling from 2 to 6 inches or more in diameter. Fistulous openings and granulomatous swellings are usually present on the surface, and the skin is adherent to the bone. The pus from actinomycotic fistulae and abscesses of the bone and soft parts of the head is thick, creamy or gummy, and yellowish or white in color. That even small lesions are painful in the early stages is shown by violent objection of the cow to examination. Thus the course, as measured by the rate of growth of the swelling, is

extremely variable. In certain groups the ostitis takes on a malignant form that soon destroys, while in others the chief manifestation is an unsightly blemish. After the swelling ceases to grow, the fistulous openings and granulations heal, but the lesion may become active again after a few months. So long as the lesions remain small and circumscribed there is no disturbance of nutrition, and even when they become several inches in diameter the general condition may remain good. Chewing is painful when the alveoli are involved in an extensive rarefying ostitis of either the upper or lower jaw.

The prognosis is not good, for the bony growth cannot be removed surgically, neither can it be resorbed. At best the affected animal has a greatly diminished value. In herds under municipal dairy and milk inspection, such animals are not permitted to remain. While there is no evidence of direct conveyance from animal to man, or between animals, the possibility that such conveyance may occur justifies isolation or slaughter. The cyclic recurrence of the disease in an enzootic form does not completely harmonize with the theory that it is a wound infection disease. In a group of young heifers the disease may attack the bones of the face in 10 per cent of the individuals during a period of six to twelve months. Following this there may be an interval of freedom from the disease on the farm for four or five years, when another similar outbreak may occur; this is suggestive of contagion.

Actinomycosis of the soft parts of the head and throat presents a variety of symptoms according to the location and extent of the disease. In the *subcutaneous tissues* of the submaxillary and parotid region one finds swellings that are circumscribed, diffuse, or multiple. On palpation they are neither hot nor painful and usually are not attached to the bone. The fact that they are movable distinguishes this type from genuine actinomycosis. In some cases there is a marked swelling of the adjacent lymph glands. The most frequent location is in the submaxillary region and near the lower border of the parotid region. Occasionally there may be a firm, circumscribed, round subcutaneous nodule on the face about midway between the eye and the commissure of the lip.

Actinomycosis of the tongue is marked by salivation and difficult mastication. On palpation along the lateral border, ventral to the papillae, one may feel submucous nodules, especially along the posterior half. The tissue in this part is firm to the touch—"woody," and manipulation of the tongue is painful.

Actinomycosis affecting the pharyngeal ring is less frequent than the preceding forms, but it is important in relation to tuberculosis of the retropharyngeal or submaxillary lymph glands. In both affections there may be an inspiratory dyspnea. This symptom, when chronic, is com-

monly taken as positive evidence of tuberculosis of the retropharyngeal lymph glands. But actinomycotic granulations or abscesses in the pharynx or larynx, or actinomycotic abscesses in the submucosa of the pharynx may induce symptoms identical with those of tuberculosis of the retropharyngeal lymph glands. Abscesses outside the lymph glands or the mucosa of this region are difficult to locate, even when they are large enough to induce inspiratory dyspnea.

The *prognosis* in actinomycosis of the soft parts is good. With few exceptions drainage is possible, and the entire condition is less malignant than rarefying ostitis.

Treatment.—Actinomycosis of the subcutaneous tissue in the region of the head responds to appropriate surgical treatment. Under local anesthesia, abscesses are opened, and fibrous circumscribed growths may be dissected out. Pack the wound cavities with gauze saturated with tincture of iodine. After twenty-four to forty-eight hours these may be removed and open wound treatment employed. After the wound is nearly or quite healed there may be a gradual return of the swelling because of incomplete elimination of the infection. In such a case inject Lugol's solution (5 to 20 cc.) directly into the swollen tissues. The immediate effect is a marked increase in size, but the growth is finally resorbed and ceases to be active.

Potassium iodide exerts a specific curative effect in many cases of actinobacillosis. The daily dose is from 1.5 to 2.5 drams (6-10 Gm.). If iodism appears suspend the treatment for a week. The symptoms of iodism are lachrymation, marked dandruff, and loss of appetite. In addition, iodine may be applied locally in the form of tincture of iodine or Lugol's solution. The internal administration of iodine is of doubtful value in extensive involvement of the bone. At several points around the base of a swelling, Lugol's solution of iodine may be injected subcutaneously, as well as directly into the diseased tissues. Beneficial effects from iodine have been chiefly observed in involvement of the soft parts. I have administered potassium iodide repeatedly in actinomycosis of the bone with no apparent benefit. If the bony growth is small and not growing rapidly, its development may be retarded by combined local and internal medication with iodine. After the development has ceased it may be possible to secure healing by means of an operation on circumscribed small bony growths attached to the lower jaw. Sodium iodide intravenously (30 Gm. per 500 cc. water) at 18-day intervals has proved effective and convenient in the treatment of actinomycosis of the soft parts, not involving the bone.

In human medicine vaccination has been employed in a few cases with apparent benefit.

Various forms of iodine have been recommended for their special effect upon actinomycotic growths; some of these may be administered intramuscularly or intravenously. There is no evidence that their action is different from that of the ordinary forms of iodine. In an experience limited to the treatment of ten cases, Kingman and Palen[9] conclude that "streptomycin in the handling of actinomycosis of the bone in cattle has been extremely encouraging." Dosage consisted of 5 Gm. of streptomycin in 10 cc. distilled water injected subcutaneously around the periphery of the area every day for three days.

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GLANDERS

(Malleus; Farcy)

Definition.—A chronic infectious disease of solipeds caused by *Bacillus mallei (Malleomyces mallei)*. It is characterized by the formation of nodules and ulcers in the lungs, the skin (farcy), and the nasal mucosa. Infrequently man is infected.

History.—Glanders was described by Vegetius as early as 400 B.C. It received the Greek name "malleus" from Aristotle. Until about the period of World War I it continued to be an important disease among horses. Recognized as contagious as early as the fourth century, control measures somewhat checked its spread in peace times with the exception of a few years early in the eighteenth century when authorities at the Alfort Veterinary College denied its contagiousness. Its greatest prevalence has attended wars and the periods immediately following,

when conditions favor the distribution of infected animals. Thus the disease has chiefly prevailed where horses were assembled in large numbers, as in armies, cities, and camps. With the scattering of horses from such centers it spread to the country. Effective methods of control under war conditions were not found until the period of World War I. For centuries the disease has been recruited in Europe from Russia where a primitive animal industry prevails. In 1912 there existed a chain of diagnostic laboratories extending from Budapest to Copenhagen, and in all of these blood was examined for the detection of glanders in horses entering from Russia.

Etiology.—*Prevalence.*—With increasing knowledge of the use of mallein and the passing of horses from the cities, glanders has become almost unknown in this country. Germany and other countries in Central Europe suffered from a heavy invasion during and after World War I, but the disease is now greatly restricted except in those countries where sanitary control is undeveloped.

Man may acquire equine glanders by direct or indirect contact with diseased animals, though this form is relatively infrequent. Artificial inoculation is successful in guinea pigs, rabbits, dogs, goats, camels, mice, and rats. Sheep, swine, and pigeons show a high resistance. Cattle may be infected artificially, but they never contract the disease by natural infection. Osler states that among laboratory workers *Bacillus mallei* has caused more deaths than any other germ, and that in working with it greatest precautions should be taken. Under favorable conditions horses may recover.

Bacillus mallei is a non-motile, straight or slightly curved gram-negative rod, 2 to 5 microns in length. The ends are usually rounded and the contour irregular. Its habitat is in the nasal secretions and in nasal, pulmonary, and skin lesions of glandered horses. In stained smears from lesions it is ordinarily not recognized, for there is no specific stain. On potato cultures it often gives a honey-colored deposit. When taking specimens for culture (lymph glands, abscesses) they should not be incised. It survives for not more than two to three months outside the body.

Modes of Infection.—Transmission from diseased to sound animals is usually through the medium of discharges from the lungs, nose, or skin. These contaminate water pails, mangers, troughs, harness, utensils, food, bedding, etc. The port of entry usually is intestinal, though it may occur by inhalation or through the abraded skin. Carnivorous animals are infected by eating infected raw horse meat. Man is infected in a similar manner, or in the laboratory, or infrequently in stables through abrasions in the skin or mucosa. Infection through wounds or by in-

halation is rare. Where many horses are together, as in camps and armies, conditions favor the spread of glanders through common troughs and mangers.

Types of Infection.—After entering the digestive tract in contaminated food or water, infection is carried from the pharyngeal or intestinal mucosa in the circulatory system to the lungs. Here it localizes and forms the primary lesions. The presence of infection in the circulation is shown by fever immediately following artificial feeding. While a few authorities regard the intestines, spleen, and liver as the primary seat of the lesions, the majority hold that the primary development is in the *lungs*. This belief is supported by the findings of marked changes in the lungs of many horses not otherwise manifestly diseased. In addition to the primary lesions in the lungs, the infection tends to localize in the nasal mucosa (*nasal glanders*) and in the skin (*farcy*).

Morbid Anatomy.—*Lung Lesions.*—The most constant lesions are in the lungs. They contain tubercle-like nodules (nodular glanders), or there may be a lobular pneumonia (diffuse glanders). Often the nodules are extensively scattered; they are grayish externally, yellowish in the center and surrounded by an inflammatory zone. The pneumonic type presents larger brownish red nodules, up to ¼ inch in diameter, and these may have fused to form a large granulomatous mass.

Nodular lung glanders may be acute or chronic. The two forms have been described as follows by Frothingham:[1]

The acute nodule presents small dark-red foci of hemorrhagic pneumonia; in size it varies from 1 to 4 mm. In a more advanced stage the nodule is gray with a yellowish center and surrounded by a red zone—Fig. 95. *The chronic nodules* may be few or many, as in the acute form. The pneumonic focus becomes organized by the formation of epithelioid cells, giant cells, and connective tissue, as in a tubercle. There is a central necrosis with some calcification; surrounding this is the epithelioid region, and next a connective tissue wall, beyond which the lung is normal—Fig. 96.

In horses that have been condemned and slaughtered because of reacting to the mallein test, there have been small lung nodules which have caused dispute as to whether they were glanders or parasitic. The question is readily answered by microscopic examination of a histologic section. Parasitic nodules are more uniform in structure and bluer in color when stained with hematoxylin for they are composed largely of lymphoid cells. Eosinophiles are plentiful, while they are absent or rare in a glanders nodule—Fig. 97.

The diffuse form may also be acute or chronic. Lung glanders is usually chronic.

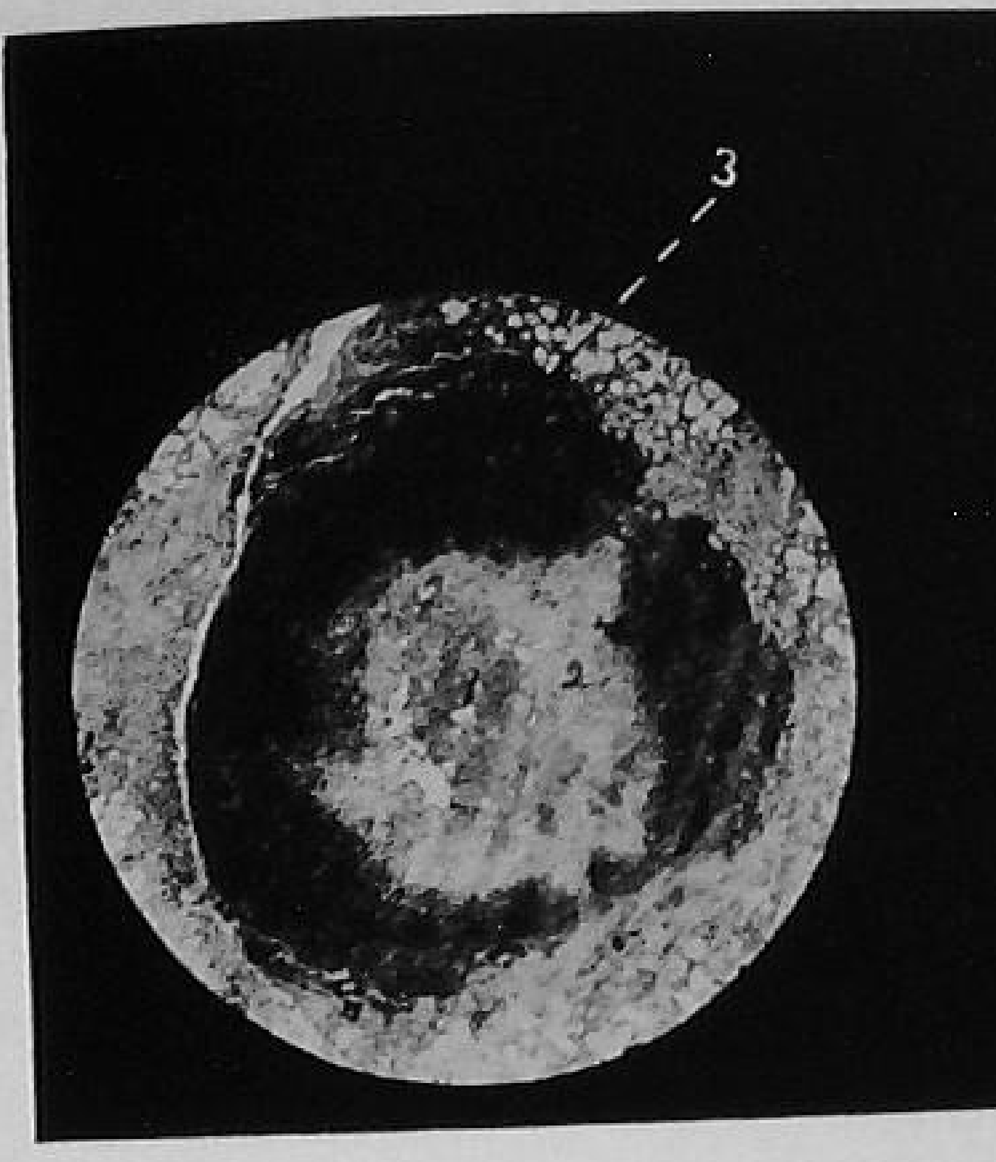

Figure 95.

Figure 96.

Fig. 95.—Acute glanders nodule. 1. Leucocytes and nuclear detritus. 2. Pneumonic area. 3. Hemorrhagic zone (× 30)—Frothingham.

Fig. 96.—Chronic glanders nodule. 1. Central necrosis. 2. Epithelioid zone with remains of alveolar walls. 3. Connective tissue capsule (× 30)—Frothingham.

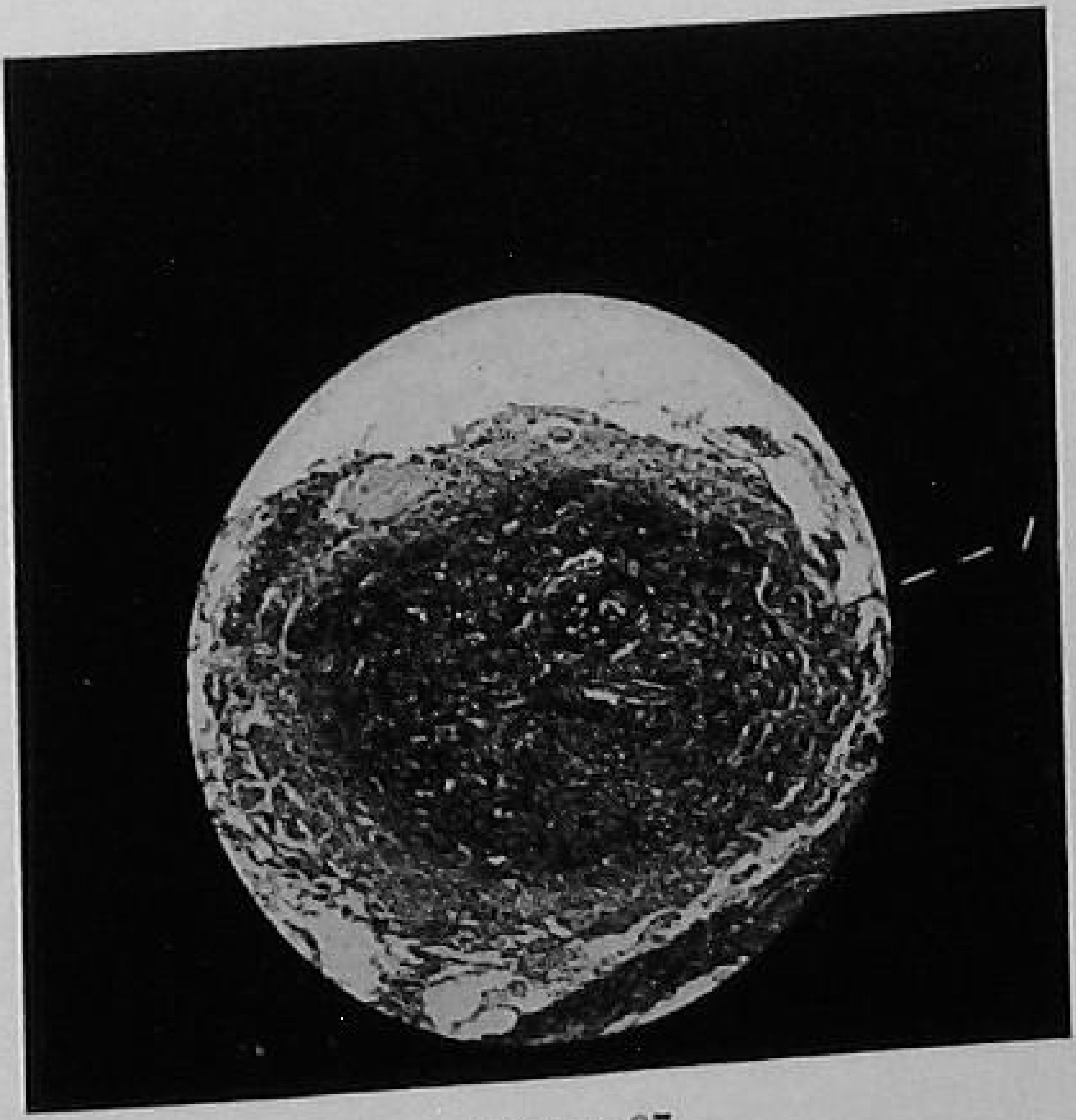

Figure 97.

Fig. 97.—Parasitic nodule. Near the center is a portion of a parasite 1 (× 40)—Frothingham.

Skin lesions present nodules from 1/4 to 1/2 inch in size; the center is soft and may contain honey-like pus. Beneath the skin are larger nodules and abscesses. The essential lesions are granulomatous masses that tend to break down and form ulcers. The ulcers present thickened irregular margins, and a glistening red surface covered with reddish or yellowish pus. The lymphatics are swollen and firm and along their course may be small typical glanders abscesses.

Nasal lesions, when recent, present gray yellowish nodules 1 to 2 mm. in diameter; the surrounding mucosa is red and swollen. When these nodules first break down they leave round ulcers. Fusion leads to the formation of large ulcers with irregular thick-walled margins. The surface is yellowish and glistening. Healed ulcers leave scars which are irregular and present radiating margins; they may be extensive over the turbinated bones. Similar changes may be found, though less frequently, on any part of the respiratory mucosa—larynx, trachea, bronchi.

Lymph glands adjacent to glanders foci, such as the submaxillary, peribronchial, axillary, and inguinal, are often the seat of glanderous lesions. In acute types these are prominently swollen; in chronic forms they are adherent to the surrounding tissue and contain pus foci.

Other organs which may contain lesions of glanders are the mucosa of the digestive tract, the liver, spleen, kidneys, testicles, brain, spinal cord, and bones.

Symptoms.—The period of incubation varies according to the intensity of the infection. There may be a rise in temperature, and local lesions may occur within three to five days after artificial inoculation. Balint has reported a case of natural infection after an incubation period of only seven days. After natural exposure the incubation period is several weeks and it may be months.

Chronic Pulmonary Glanders.—Chief interest attaches to chronic pulmonary glanders, for horses affected with this type often spread the infection without themselves showing symptoms. When a case of latent lung glanders is added to a group of horses or mules in a corral, infection may soon become general through contamination of the mangers and troughs. Systematic temperature readings may disclose a recurrent fever, and blood examination an increase in leucocytes. The initial physical signs are an unthrifty condition and heave-like symptoms.

Chronic nasal glanders first shows as a lymph-like unilateral nasal discharge. With the development of ulcers the discharge becomes purulent and flecked with blood. Inspection of the nasal mucosa may reveal typical ulcers or nodules; unilateral swelling of the submaxillary lymph glands is the rule. A recent glanders nodule is round, grayish or grayish

red, somewhat transparent, about 1 mm. in diameter and surrounded by a red zone. Within a day or two this may become yellowish and purulent. Glanders ulcers are deep, crater-like, with prominent margins and a fat-like base. They fuse to form large ulcers with irregular margins. Healed ulcers present a radiating or star-shaped cicatrix. General symptoms are often present in the form of poor condition and recurrent fever. Chronic skin glanders is usually associated with the nasal form.

Chronic skin glanders, like nasal glanders, is usually metastatic from the lungs. It is characterized by nodules (farcy buds) which often appear rapidly under the skin and soon degenerate into abscesses and ulcers. The cutaneous nodules are ¼ inch (6 mm.) in diameter and soon they are transformed into superficial or deep ulcers; often they are joined by swollen lymph vessels. The deeper subcutaneous nodules are larger—1 to 1.5 inches; they may become encapsuled, or rupture and leave fistulous tracts. The pus in a skin abscess is about the consistency and color of dark honey; it is characteristic of *B. mallei.* The most frequent location of skin glanders is on the medial surface of the hind limbs, but it may appear anywhere.

Acute glanders begins with chills and a high fever—106° to 108°F. It is characterized by rapidly spreading ulcers and suppuration on the nasal mucosa. The nasal discharge soon changes from mucopurulent to hemorrhagic and ichorous. There is a marked swelling of the submaxillary lymph glands. Inspiratory dyspnea may result from edema of the glottis. Soon there follows secondary acute skin glanders with the formation of nodules, abscesses, and phlegmonous swellings of the limbs. Death occurs within a few days, sometimes within a week. This is the usual form in the ass and mule.

Course and Prognosis.—Acute glanders leads to death within a few days and the symptoms are characteristic. Chronic latent glanders often is disclosed only on mallein or serological tests. In pastured or range horses recoveries are frequent, and these animals are dangerous sources of infection.

Diagnosis.—Since glanders has become relatively rare in the United States, its diagnosis is entirely a problem of the detection of latent pulmonary forms. It may be encountered only among horses exposed in transportation or fed and watered in large groups.

Mallein Test.—Mallein, like tuberculin, was first used subcutaneously as a diagnostic test. A reaction consisted in a post-injection fever, often asociated with swelling at the point of injection. The subcutaneous mallein test is no longer used, and in Germany it is prohibited because of interference with serodiagnosis.

The ophthalmic mallein test displaced the subcutaneous method. It is

more accurate, more easily applied, can be frequently repeated, and does not interfere with serodiagnosis. It is especially useful where a prompt decision upon thousands of horses is required. Animals under test should be rested and free from influenza, colds, or other conditions affecting the conjunctiva. Instillation of mallein into the lid-sac of a normal eye causes a specific purulent conjunctivitis; this appears in not less than 3 to 6 hours, reaches its height in 8 to 12 hours, and lasts from 1 to 2 days. Two or three drops are placed in the left lid-sac, using a small dropper or a glass rod. The instillation is made in the late afternoon or evening. The horses are then tied short to avoid loss of exudate by rubbing. They are fed hay at night, but not thereafter until observations have been completed. The first observation is made in the morning, after about twelve hours. Two other readings may be made at 6-hour intervals. The results are interpreted as follows (*Army Regulations* 40, 1921):

Eye unchanged	Negative.
Mucous discharge	Suspicious.
Conjunctivitis any degree with or without mucous discharge	Suspicious.
Mucous or mucopurulent discharge with purulent flakes	Positive.
Purulent discharge	Positive.
Purulent discharge with edema or gluing together of the lids	Positive.

According to German writers a serous, or seromucous exudate with no pus, a grayish white exudate, or clumps of mucus constitute a negative result.

It is claimed of this test that it reveals 90 to 100 per cent of latent glanders. So long as one latent spreader remains in a group the disease will continue to spread, and the period of incubation may be short.

The Intradermic Test.—In 1915 Captain Goodall[2] of the South African Veterinary Corps reported the superiority of the intradermic test. He observed that while the ophthalmic instillation of mallein yielded fairly good results, it had the following grave drawbacks: "Often the reaction is transitory and may be easily missed; a small discharge may be found in the eyes of normal animals; animals or attendants easily rub away the discharge." The intradermic test gives distinct reactions in animals that may repeatedly have passed the ophthalmic test. Because of this superiority it has become the standard test in the United States Army. The introduction of this test made it possible to remove the scourge of glanders from the army horses. The test consists in the intradermic injection of 0.1 c.c. of concentrated mallein into the skin of the lower eyelid. Injection is made with a 1 cc. Luer-type glass syringe that has been sterilized by boiling. Immerse the needle in alcohol after each injection. Avoid the injection of animals

showing evidence of irritation of the eyes. The injection is made into the skin about one-fourth inch from the margin of the lid, midway between the inner and outer canthus and parallel with the lid. Injection is usually made in the right eye; this permits the resting of the right hand against the head in order to follow any movement of the animal. A transient swelling may appear within two or three hours, but the specific reaction is not observed until the end of forty to forty-eight hours. It is in the form of a marked edematous swelling, often of both lids. The lids may be nearly or completely closed. In addition there may be a purulent conjunctivitis, photophobia, and depression; it is a combined eye and skin test. The reaction may appear before the 48th hour and it may persist for three or four days. Slight puffy swellings limited to the lower lid or extending somewhat lower have no significance.

The complement fixation test is the most accurate method of serodiagnosis and it has been widely used. It may fail to reveal 5 to 10 percent of infected animals. On the other hand, reactions may result from other infectious diseases, such as strangles, influenza, and purpura, from anemia, during convalescence, pregnancy in mares, and in the normal blood of the mule and ass—Zwick.[3]

Subcutaneous or intraperitoneal inoculation of guinea pigs with suspected material, usually pus, causes orchitis in the male. At the seat of inoculation there develop typical swelling, abscess formation, and ulceration. Abscesses may form along the adjacent lymph glands. Infected guinea pigs die in from three to four weeks; some may live two to four months, and they may recover. Only a positive result is conclusive, for only 20 to 25 per cent of guinea pigs inoculated with fresh glanders material contract the disease.

Bacteriological examination of stained smears from suspected material has little diagnostic value.

Control.—The control of glanders has been accomplished by repeated application of the mallein test (every two to three weeks) and the destruction of all reactors. Where many animals are involved, it is an advantage to distribute them in small units. Mangers and troughs used by infected animals need to be thoroughly cleansed and disinfected.

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EPIZOOTIC LYMPHANGITIS

(African Glanders; Pseudoglanders)

Epizootic lymphangitis is a specific lymphangitis of horses and mules caused by the fungus *Saccharomyces (Blastomyces) farciminosus*. Smears from pus yield opaque, yeast-like cells 3 to 4 microns long by 2.5 to 3.5 wide. This disease is frequent in Europe where it assumed considerable importance during the World War I. It is reported as highly prevalent in Japan, China, and South Africa.

The *mode of infection* is through wounds and abrasions of the skin. Infection is carried by bedding, utensils, harness, and possibly by flies.

Symptoms.—After an incubation period of 6 to 8 weeks, the lymph vessels near the port of entry present nodular swellings followed by abscess formation. The usual location is on the hind limbs in the vicinity of the hocks, but lesions may appear anywhere on the body or limbs. Following the appearance of nodules and abscesses the adjacent lymph vessels and lymph glands become swollen and swelling may become extensive. Occasionally nodules and ulcers appear in the nasal mucosa. The course is over a period of months. Emaciation is frequent and the mortality is from 10 to 25 per cent.

In differential diagnosis one considers glanders, ulcerative lymphangitis, phlegmon with abscess formation, and contagious pustular dermatitis. The fungus may be recognized in smears from the pus.

Treatment.—Recovery occurs only when the lesions receive early treatment. The affected tissues are removed surgically followed by cauterization. Old and extensive lesions are incurable. The most effective method of control is slaughter followed by disinfection of the stable.

BRUCELLOSIS IN CATTLE

(Contagious Abortion; Bang's Disease)

Definition.—Contagious abortion of cattle is a widespread destructive disease chiefly affecting dairy breeds and caused by *Brucella abortus* (Bang's bacillus). *Pathologically* it is characterized by inflammatory and degenerative changes in the pregnant uterus, the fetal membranes, and the fetus. Following expulsion or death of the fetus various degrees of metritis persist. Occasionally this leads to early death of the cow from acute septicemia, or to a septic condition that leaves her severely damaged because of extensive lesions in the genitals, arthritis, and general disturbance. More often, the lesions are confined to the uterus, causing a temporary, intermittent, or permanent sterility; with few exceptions these lesions may be recognized on the placenta at

each subsequent parturition. The characteristic immediate *symptoms* are expulsion of the fetus with or without systemic reaction. The chief subsequent manifestations are sterility and a diminished milk flow due to premature lactation. While the symptoms and lesions of Bang's disease are characteristic they are not pathognomonic, since identical phenomena may be caused by other infections. In its epidemiology, however, the rapid spread is rarely duplicated by any other disease.

In the male, *Br. abortus* may cause abscess formation in the testicle and the epididymis.

According to certain authorities *Br. abortus* is not pathogenic to adult cows; the cow plays a passive role, the morbid changes being confined to the fetus and its membranes. This conception ignores the fact that identical lesions are present in the fetal and maternal placentae. It is based on the theory that secondary infection, not *Br. abortus,* is responsible for the attendant metritis and other lesions in the dam. Assuming the accuracy of this view, medical authorities do not exonerate primary invaders for damage done by secondary infection. Regardless of one's view on where one infection leaves off and another begins, the chief loss from this disease is in the dam.

In this connection the original conclusions of Professor Bang[1] are of special interest: "This discovery indicates that the epizootic abortion ought to be regarded as a specific uterine catarrh, determined by a definite species of bacterium. It is true that the uterine mucous membrane was not strikingly altered, but a chronic catarrh is not necessarily associated with striking anatomical alteration. In my opinion the very abundant exudate, which contained a quantity of shed epithelial cells, pus cells, and detritus, must necessarily have been furnished by the uterine mucous membrane, and not by the thin chorion, and consequently the disease must be regarded as a uterine catarrh."

Etiology.—*General Prevalence.*—Contagious abortion of cattle has a world-wide distribution, and with few exceptions Bangs's bacillus has been carried wherever improved breeds have been introduced. Thus it is widely prevalent in all of the intensive dairy districts of the United States and of every other country with the exception of the Channel Islands. Not only is contagious abortion widespread, it is intensively distributed. Most dairy herds of average size are infected at one time or another. In sections where the raising of cattle for sale is an important business, and where the interherd change of cattle is unimportant, the intensity of the infection is probably less. Yet in these localities it sometimes becomes so widespread that it is little short of an epidemic. After an extensive outbreak it gradually recedes, but the majority of herds suffer continuously, especially from abortion in first-calf heifers.

When isolated from neighboring cattle a herd may escape infection for a time. The disease has prevailed for many years and the losses therefrom are beyond computation. Under the current control program of blood testing of herds and vaccination of calves, brucellosis in dairy cattle has been greatly reduced. As reported by McAuliff,[2] the inclusion of two-thirds of the cows and calves in New York under this program in 1945-50 has practically eliminated storms of abortion. As published in the 1952 report of the U.S.B.A.I.,[3] the federal program for the previous year was as follows: blood agglutination tests of cattle, 7,491,327; per cent reactors, 4.2; calves vaccinated, 3,179,251; brucellosis-free areas (not more than 1 per cent), 345 counties in 18 states, Maine, North Carolina and New Hampshire. Brucellosis ring tests were applied to 454,732 herds of which 135,967 were positive; use of the test eliminates temporarily the requirement of blood testing in negative herds. An increase in number of reactors, in comparison with 1951, was due to an intensive program in Wisconsin, where the percentage of reactors was 10.3 of 1,469,320 cattle tested.

Age.—When the disease becomes active in a negative herd, females abort irrespective of age. After the first or second years, when the survivors have established a relatively strong resistance, it prevails chiefly among the first-calf heifers. A high percentage of these may abort each year. Animals less than one year of age are said to be only slightly susceptible and rarely acquire permanent infection. This opinion should not be accepted too literally. I have repeatedly observed that where large groups of heifer calves are raised on infected milk, a few of the individuals are high reactors and infected at breeding age, and following conception they usually abort.

Immunity.—As in most infectious diseases, a few individuals resist all exposure and never contract the malady. Another small group abort and never conceive thereafter. A third group abort repeatedly and conceive with difficulty. A fourth group include what Birch terms "ordinary susceptibles." They abort once or twice, pass through an unstable breeding period, become relatively tolerant, and carry their calves to term; this is by far the largest group. The immunity thus obtained is expensive, and there is no way of knowing in advance that even this will be accomplished. The term immunity, as applied to this disease, is somewhat misleading. If the cow becomes a "regular breeder" and carries the fetus "to term" she is said to be "immune." Observations covering the lives of these immune cows, especially the high reactors, show that even though they have ceased to abort frequently, the breeding regularity alone is 20 per cent below that of negative cows on the farm, when measured by the time and services required for conception

after each calving and by the number of live calves. A useful formula for comparison is to credit a cow with "A" for each conception within six months after calving, and for each live calf born at 265 days or more; exceptions are given a "B." After a cow has aborted, chief interest relates to the damage her tissues have already acquired, not to the immunity she may possess against additional damage. The disease is so inconstant, due to a variable resistance of the animal, a variable intensity of the infection or variations in methods of feeding and care, that an accurate estimate of the injury to an individual cow is difficult to determine before the end of her reproductive life. A group of negative cows that were sickly and unthrifty as calves, and that are handled with no regard for genital hygiene may be less productive than a well-grown thrifty positive group.

Bacteriology.—*(a) General Character.*—*Brucella abortus* is a small gram-negative rod 1 to 2 microns in length by 0.5 micron wide. In smears from the uterine exudate or chorion it appears in the form of dense clumps giving the appearance of cocci. The characteristic appearance of the bacilli in uterine exudate was described by Bang[1] in 1897 as follows: "The examination of a cover-glass preparation made from the yellowish exudate and stained with Loeffler's methylene-blue immediately showed the presence of a small bacterium, apparently in pure culture. This organism was present in very considerable numbers; many examples lay free, but most striking were the large dense clumps of bacteria. Closer examination showed these heaps were included within cells, whose bodies were in this way greatly distended. . . . In the dense heaps the bacteria had mostly the appearance of cocci, but some of the free-lying individuals were of longer shape, and these were at first regarded as short oval structures; closer examination, however, under very high magnification showed that we had in fact to deal with a small bacillus."

The *vitality* of *Br. abortus* was also recognized by Bang, who observed that the bacilli remained alive at least nine months in the uterus of a cow after the death of a fetus without expulsion, and at least seven months in a tube of agar-serum in the ice chest. Experiments conducted by Cameron[4] to determine the resistance have shown that it will survive 114 days in tap water kept at a temperature of 24.8° F.; 100 days in bovine feces which were kept moist in an unheated cellar; 120 days in bovine feces kept in test tubes in a laboratory cupboard and dried slowly; 66 days, the maximum interval covered, after having been in a wet soil stored in an unheated cellar; 4.5 hours when exposed to direct sunlight; 30 days when kept in burlap sacking in an unheated cellar; and 121 days, the maximum interval covered, when dried in the pres-

ence of nutrient material. It is obvious that the organism has a relatively high resistance outside the body and that its capacity to survive for months under usual pasture or stable conditions must be met wherever control measures are in operation.

(b) Distribution in the Body.—In the *uterine exudate, fetal membranes,* and *fetuses* of aborting cows, the bacillus is found in great numbers. In smears from the chorionic surface of affected membranes, especially from the margins of the leathery thickenings, the bacilli are found both free and enclosed in epithelial cells. Often the bacilli are present in the fetal membranes and uterine exudates of infected cows after delivery of a normal calf at term; such cows may have aborted at previous parturitions, or they may have first acquired infection late in the current pregnancy. In infections late in pregnancy the bacillus has been recovered in the fetal membranes before the appearance of agglutins in the blood, and as early as one month after exposure to natural infection. Where routine examinations of all placentae from a recently infected herd are made, and guinea pigs are inoculated, as high as 50 per cent of the infected placentae may be free from gross lesions of Bang's disease and come from cows that calve at term. Occasionally bacilli may be found in the placenta of a cow that calved at term, and that did not subsequently give a positive blood reaction. In one such case, in addition to reproducing the disease in a guinea pig, the placenta presented a gross appearance of infection in the form of leathery areas. In the pregnant uterus the bacilli live and multiply, chiefly in the epithelium of the embryonal chorion. They enter the fetus in the swallowed amniotic fluid and are found in the fetus in the digestive system and lungs.

Routine examination of uterine exudate fails to reveal the organism in the *uterus* for more than two months following the act of abortion, and this has led to the inference that it does not remain here for a longer period. However, in two cases in our clinic it was recovered from the nongravid uterus four and five months after abortion; guinea pigs were infected by means of inoculation with uterine tissue. Birch and Gilman[5] have reported three cases from which the uterus yielded *Br. abortus* over a year after the last previous abortion; guinea pigs were successfully inoculated with uterine tissue. These observations suggest that the infection may remain indefinitely in the uterus. Vaginal mucus from infected unbred heifers in estrum is free from the organism, according to observations made by Thomson.[6]

In the *udder,* infection is harbored indefinitely, but it does not cause perceptible injury to the glandular tissue; this organ is regarded as the chief permanent habitat of the bacillus. Its presence here is usually

associated with a relatively high blood titer. Gilman[7] has reported that in his work "in no instance was the organism recovered from the milk of a cow with a lower blood titer than partial at 1:320. . . . Others have reported exceptions to this, but they are quite unusual, and are by no means a disturbing factor." On this subject Cotton and Buck[8] state, "The results of these tests, in general, have confirmed those of earlier work which were to the effect that no *Br. abortus* infection was found in milk of cows reacting to a titer of 1:100 or less, but that this organism was present in about 86 per cent of those cows having blood titers of 1:200 or above. . . . Our experience leads to the belief that the blood test, when using titers of 1:200 and above, as the test is made at the Experiment Station, are considered as denoting udder infection and those of 1:100 or less as denoting freedom from infection, gives more reliable information regarding udder infection than testing milk from individual quarters for agglutins."

In an article on vital statistics by Udall, Cushing and Fincher,[9] the milk of four cows is reported as positive from animals whose blood reacted at from competely negative to partial at 1:60. Gwatkin[10] also reports that cows with low blood serum reactions may have infected udders. He concludes that "the serum titer is not helpful in determining whether or not the placenta or udder is likely to be infected," and that "the udders of these cows may play a more important part in the dissemination of infection than has usually been attributed to them in recent years, and unfortunately the serum titer is not a guide as to whether or not udder infection is present."

Observations by Olaf Bang and Bendixen[11] on 20 cows whose milk contained *Br. abortus,* showed that 85 per cent would be included in cows reacting at 1:100; three of the 20 failed to react at this titer.

When a cow becomes infected the bacillus seems to be harbored indefinitely. While its favorite location is the udder and genital organs, it may be found in the *lymph glands, spleen, bones,* and *joints.* In the male it may be found in the *testicles, seminal vesicles,* and *epididymes.* Boyd[12] and co-workers have reported its presence in *carpal hygromata* in cows.

Br. abortus has been found repeatedly in the male genitals. Schroeder and Cotton found it in the epididymes, while Buck and Creech[13] obtained it from the seminal vesicles. According to the Federal Bureau reports, it is found in the semen in 10 per cent of reacting bulls.

(c) The Bacilli outside the Body.—From the observations of Bang,[1] Cameron,[4] and others it is obvious that the ability of Bang's bacillus to live outside the body under various conditions of moisture and temperature favors its conveyance to susceptible animals. There is

little direct knowledge of its occurrence in *mangers and drinking places*, as in the case of the tubercle bacillus, but there is sufficient reason to suspect premises and carriers, as in tuberculosis and other infectious diseases. *Milk* from infected herds practically always contains the bacillus. Milk from infected cows is of special significance from a public health standpoint, since it is a source of brucellosis in man. The capacity of the cow to carry all three types of infection, to contaminate the milk, and to transmit the infection to man, is responsible for the present demand by public health officials that all milk be pasteurized, and even that only cows that are negative to the agglutination test be qualified for market milk production. The chief sources of infection outside the body are uterine exudate, placental membranes, an aborted fetus, a live newborn calf from an infected uterus, raw milk, and cheese made from raw milk.

Artificial Transmission.—Vaginal injection of pure cultures causes abortion in pregnant susceptible cows in from five to ten weeks. Infection is readily produced by subcutaneous or intravenous injection. Introduced into the teat canal, the bacilli appear in the fetal membranes and are excreted in the milk for weeks and months. Successful inoculation by placing suspension of *Br. abortus* in the conjunctival sac and upon the skin of cows has been reported by Cotton and Buck.[14] In guinea pigs subcutaneous inoculation of infected material causes a chronic disease with the formation of small tubercle-like foci in the lungs, liver, and kidneys, swelling of the spleen, and inflammation of the lymph glands. Pigs injected for diagnostic purposes are commonly killed for examination at the end of six weeks. Cotton, Buck, and Smith[15] have reported successful transmission of Bang's disease to pregnant cattle by the application of *Br. abortus* suspensions to a slightly abraded surface of the skin and to small areas of the intact skin.

Types of Infection.—Three types of infection are recognized: *Br. abortus, Br. suis, and Br. melitensis.* Both suis and melitensis are reported by Huddleson[16] to be involved in infections in cattle in many of the southern states. Among the more recent reports are those from Hutchings et al.[30,31] that the transmission of *Br. suis* from infected swine to cattle does occur occasionally in nature, and an infection cycle between swine, cattle and man is of considerable importance; and from Jordan[30] that "from time to time a raw milk supply becomes contaminated with *Br. suis* brought about by hogs sharing the same lot with dairy cows," and "when infection is transmited from infected hogs to the udder of one or more dairy cows, multiple cases of brucellosis are sure to result." The virulence of the bovine strain may vary from one

that is relatively harmless to a form that leaves a large percentage of the infected cows in a worthless condition.

Modes of Infection.—*The expulsion of a fetus or calf from an infected uterus* results in heavy contamination of everything with which the uterine exudate, the fetus, and the placenta come in contact. Birch and Gilman[5] have observed that recently infected cows spread the bacilli from the genitals at calving time in upwards of 75 per cent of cases, while those bearing chronic infections spread in like manner in less than 20 per cent of cases. Thus the expelled contents of the uterus constitute the most effective source of infection within the herd. The danger from this source is especially great when a recently infected cow freshens at term in an apparently normal manner, there being nothing about the event to arouse suspicion. The calf, carrying infection on the hair and in the feces, may further spread the infection when allowed to mingle with other animals. Such incidents are not infrequent at fairs and sales and other places where many animals from different sources are assembled. When delivery from infected cows occurs in the general stable or at pasture, exposure of others is almost certain. Because of the habit of cattle to lick such objects, a birth or an abortion at pasture may result in the exposure of every other animal in the field. In rented pastures, where stock is accepted without reference to age, sex, or any other condition, and where dry pregnant cows are kept, all susceptible animals are almost certain to be exposed before the end of the season. Where the drainage is favorable, infected material may readily pass from one pasture to another; it may be carried by dogs and other animals; or the cows themselves may break into pastures containing infected stock and thus be exposed. One of the chief obstacles to the maintenance of a clean herd under average conditions is exposure of stock at pasture or by purchased additions. After parturition, an infected cow may spread the infection as long as the uterine exudate continues to be discharged; and in the case of metritis this may be indefinitely.

The *bull* is generally regarded by clinicians and breeders as a source of infection, especially between herds; in many instances the bull apparently has brought the disease to clean herds. This may occur whenever he is used in an infected and a noninfected group. Whether the infection is permanently harbored in the diseased genitals of the bull, or is merely carried, is of little consequence; the essential fact is that he implants the infection in the vagina of the clean cow. This is especially liable to occur in the average herd where little attention is paid to breeding hygiene, and where cows are bred whenever they

come in heat, regardless of the condition of the genitals. One large herd attended in our ambulatory clinic was found to be entirely free from *Br. abortus;* in most animals the blood was negative in all dilutions. After a few years the herd sire was used on badly infected cows from a herd about two miles distant; the cows were led to the vicinity of the bull yard and served without entering any of the buildings. After repeated efforts, guinea pig inoculation of semen from this bull proved to be positive. Shortly thereafter abortions and positive blood reactions were found in the herd that previously had been negative. In the original report by Bang[1] he wrote, "By both these experiments we have furnished the complete proof that the bacillus discovered by us is the cause of epizootic abortion. They also prove that the mere deposition of the bacilli in the vagina can produce the disease." And also, "A farmer who owned 16 cows, among which abortion had never occurred, nine years ago allowed seven of his cows to be served by a bull at a neighboring farm where abortion had been observed for some years; all these seven cows aborted and no others. . . . I think every one will admit that in such a case the bull has been the carrier of the contagion. . . . I am fully convinced that the bull is very often the carrier of the the contagion, and in the fight against the disease it is absolutely necessary to take measures for prohibiting this danger. On the other hand I know of many examples where it is impossible to explain the infection in this way." Such incidents have been observed by others, including unreported observations in our ambulatory clinic. Yet certain workers have denied that the bull is a carrier of infection because they have not been able experimentally to duplicate such transmission. It is possible that the bull is an infrequent carrier of *Br. abortus*. But he cannot be disregarded as a possible source of infection on a basis of the negative experimental results which have been reported. Differences in the character and virulence of *Brucella* under field conditions may not be readily measured in a controlled experiment. Long before Bang's bacillus was discovered, the bull was believed to transmit the infection. In artificial breeding units a bull carrying a blood titer of 1:50 is disqualified for service. Elimination of *Brucella* from infected bulls is most active during a period of acute inflammation. In 37 reacting bulls, infection was found in the genital organs of 4 by Buck and Creech.[13] They concluded that infection is most apt to be found in bulls giving a high reaction to the agglutination test. It is a frequent observation that a bull may serve reacting cows over a period of years and remain negative to the agglutination test.

Age.—Experimental workers have given a false sense of security in stating their observations that animals under one year of age are rarely

permanently infected. Experience has taught that such infection is not rare. When 20 to 30 heifers are raised together on infected milk, some member of the group is quite apt to carry the infection through to breeding age, and to abort following conception. Unless the blood of such individuals is examined before breeding, an infected animal may

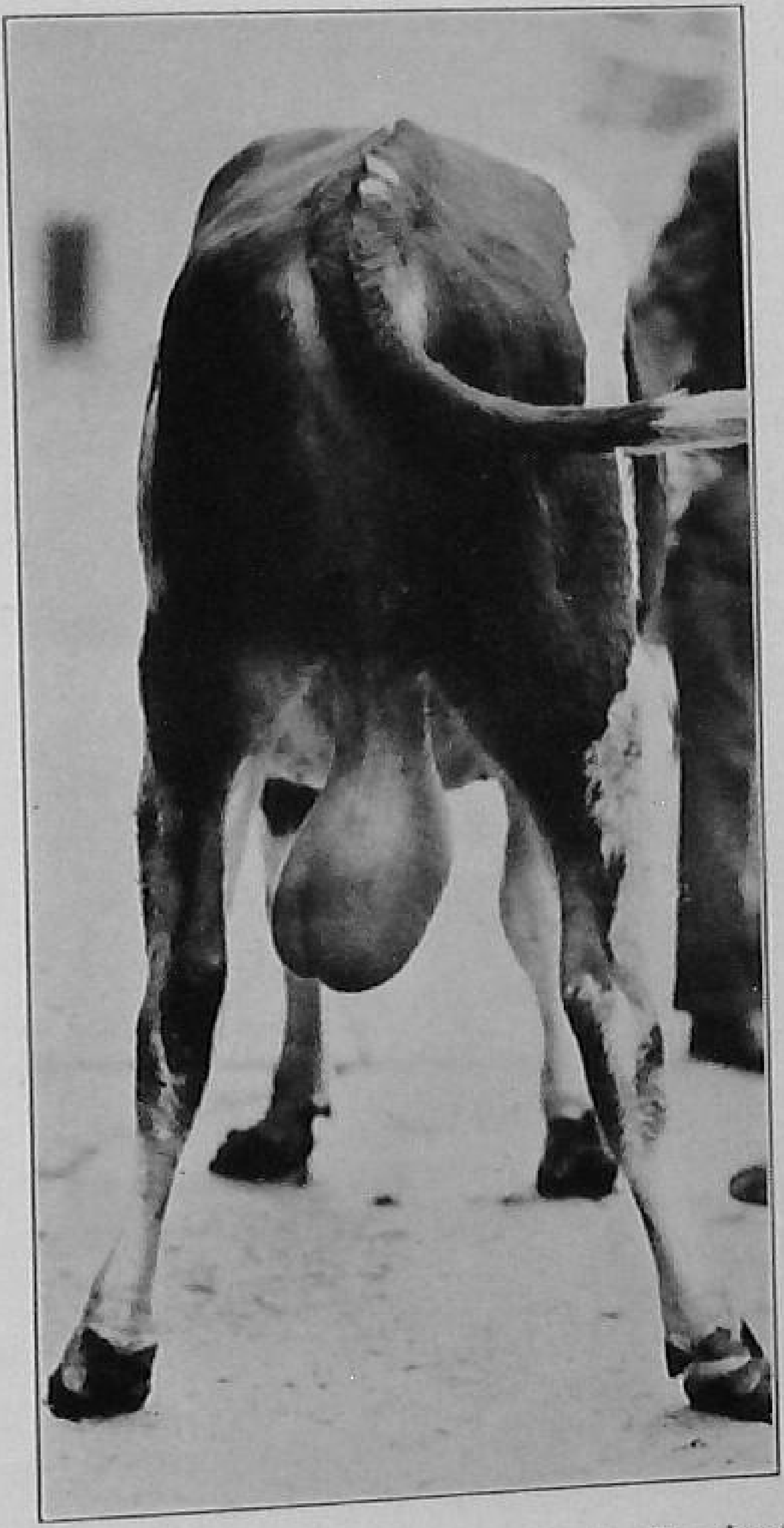

Fig. 98.—Abscess of the testicle due to *Br. abortus.*

abort and expose all of the others. The transmission of infection through the teat canal by means of milk carried from an infected udder on the hands of the milker seems possible, but there is little evidence that the disease is spread in such manner. Most owners disregard entirely the possibility of milk as a source of infection among animals.

Purchased additions are a prolific source of disease. When infected new additions are made to a herd where infection already prevails, it

is commonly assumed that no additional exposure has been acquired. On the contrary, the new addition may bring in a strain of *Br. abortus* that is more active than the one already present and thus initiate a more destructive form of genital disease. In the purchase of infected additions one also takes the risk of introducing other communicable affections of the reproductive organs; for cows with diseased genitals may harbor infections that are far more destructive than *Br. abortus.* Since cows that do not react to the blood test or abort may carry and spread infection, it is difficult to keep a herd negative if cows are added by purchase.

Pathology.—In the article by Bang,[1] in 1897, he described as follows the appearance of an infected pregnant uterus: "The external surface of the uterus was normal. Between the mucosa and the fetal membranes was an abundant odorless exudate—a dirty yellow, somewhat thin pultaceous material, of a slimy, somewhat lumpy character. At some places where the fluid constituents had run out the exudate was of semi-solid nature. . . . Nothing abnormal was noted in connection with the amniotic fluid."

An important contribution to the pathology of this disease was made by Wall[17] in 1914. He pointed out that the changes in the *uterine mucosa* are in the form of a superficial necrosis. Often there is an exudate in the lumen of the uterus, between the uterus and the fetal membranes; this may be absent or it may amount to as much as a gallon. It is odorless except when pyogenic infections are also present. It is yellowish or brown and contains polymorphs, lymphocytes, red blood corpuscles and desquamated chorionic epithelial cells. Lesions are present in both the gland mucosa and in the cotyledons. According to Wall the primary lesions are in the uterus, and the lesions of the embryo follow as a secondary result.

Ten years later Hallman[18] described his observations on the pathological changes. He does not agree with the prevalent conception that abortion disease is essentially and primarily a disease of the fetal membranes, but believes with Wall that the primary lesions are in the uterus. He writes that "in all the cases studied . . . the alterations were as well marked in the maternal placenta as in the fetal. . . . In no instance do we observe alterations of the interplacental portions of the chorion that are more extensive than those of the opposite mucosa. In most instances the alterations of the chorion are strikingly less conspicuous than those of the uterine mucosa."

The fetal membranes, then, virtually duplicate the changes in the uterine mucosa and reveal the extent of the damage to this organ. The changes in the membranes in typical Bang abortion may be duplicated

in abortion due to other causes, but in the vast majority of cases observed in our experience we are able to find lesions that positively identify this infection. The abnormal conditions are (1) thick brownish or yellowish exudate on the cotyledons and to some extent on the other parts of the chorion. (2) The cotyledons often show peripheral yellow necrosis, redness, and clumpiness of the villi. (3) Edema of the chorion is frequent. (4) A few or many of the cotyledons are missing, leaving areas of the chorion free of placental attachments. Adventitious growths are present on the interplacental chorion. (5) *There are areas of leathery necrosis on the chorion;* of the various lesions, this is the most significant. It is rare that we meet with this condition in our clinic in any other infection.

The fetus may be stained yellow with meconium as a result of intrauterine diarrhea. There may be a serohemorrhagic infiltration of the subcutis and intermuscular tissue. The body cavities may contain reddish fluid, and the serous and mucous membranes may be more or less hemorrhagic. Enteritis is frequent.

The subsequent pathological changes in the dam are inconstant. With rare exceptions the reproductive functions suffer either a transient or a permanent damage. The common statement that abortion without retained placenta leaves the cow in a normal condition, and that she has merely played a passive role, is not supported by careful observations. Even when she is left with no apparent metritis, conception rarely follows as promptly as after a normal parturition, and subsequent conceptions are often delayed or interrupted. Retained placentae with their inevitable pyogenic infections, and lesions are an important part of the disease, but the pathological retention is determined long before the cervix opens to admit other bacteria. In addition to the pathological changes in the genitals, causing sterility, one must add the diminished lactation, and the frequent metastatic arthritis that tends to localize in the hip, stifle, and tarsal joints. In consideration of these facts, it is difficult to conceive that Bang's disease is merely an affection of the fetus and its membranes, and that we may close the case with the disposal of these tissues.

Symptoms.—After infection the blood may become positive in less than two weeks and abortion may occur in about four weeks. As a rule, reactions occur in from three to eight weeks, and actual abortions are not numerous until after eight weeks. In recently infected herds neither the blood test nor the abortion are safe guides to the distribution of the infection. Cows that are infected late in pregnancy may expel an apparently normal calf and apparently normal membranes that carry Bang's bacillus as revealed by guinea pig inoculations.

In a negative herd from which we were making routine examinations of the placentae, including guinea pig inoculations, an abortion and a positive placenta were recorded in September, within one month after return to the farm from a fair. In January and February there were five positive placentae from cows that calved at term, only one of which presented macroscopic lesions; in the same interval there was one abortion. In October to December three apparently normal calvings and one abortion yielded *Br. abortus* from the placentae. In one of the normal calvings the chorion was slightly leathery and the inoculation was positive; yet the blood of the cow had not reacted above 1:100, and five months after calving it was negative in all dilutions. In five calvings and one abortion, the blood did not show a reaction until after parturition, and in two instances not until eight months later. While this may be an exceptional experience, it illustrates the possibilities in a recently infected herd.

The physical signs of abortion are of chief interest in negative herds or where a program of control is in operation. Evidence of recent abortion may be revealed by the presence of estrum in an animal known to have been pregnant. In first calf heifers, and often in dry cows, premature swelling of the udder is one of the first signs. A few hours before the act of abortion there are sinking of the sacral ligaments and the general appearance of impending delivery. With the release of the cervical seal there is a vaginal discharge of the typical abortion exudate. When the abortion occurs early in pregnancy, and even when the fetus has reached the seventh month, it may be expelled without causing premonitory signs. The presence of a dead fetus in the gutter in the morning may be the first evidence of the disease. When the death of the fetus occurs early in pregnancy, it may be discharged into the vagina and remain there for several days. Symptoms of estrum are not rare at this time. I have known a cow to be served by the bull on one day and expel a small fetus from the vagina on the following day. The fetus may die in the uterus and remain there for months, as a so-called "mummified fetus." In this case the cervical seal remains intact. Following the discharge of the fetus there is usually a discharge for from one to several weeks, and it may continue for months. Retention of the placenta with all of its complications and sequelae is frequent. In some outbreaks retention is frequent while in others it is exceptional. The effect upon the cow is more severe when expulsion of a dead fetus occurs late in pregnancy. If the diseased fetus is expelled alive it usually dies within a few hours. Birch has observed that in experimental groups infected animals are less thrifty than the controls.

Course and Prognosis.—It is commonly reported that the majority

of reacting cows finally become regular breeders. According to Birch about 50 per cent abort. Hutyra writes that the cow plays a purely passive role, and unless retention of the placenta follows abortion she is not damaged. Because of the inconstant nature of the disease, variations in breeding methods, and in hygienic precautions, an estimate of the average course and termination is impossible. Chief loss results from interruption of the reproductive functions. The degree of loss in individual cases may be determined only by the record of the reproductive life of the individual and such information is not commonly available. A survey of my own records on the entire reproductive lives of 50 infected cows in a herd containing from 100 to 150 milking animals, shows the following performance:

The records of the 50 infected cows were examined to learn the number of parturitions giving: *(a)* a viable calf at term with a normal delivery free from parturient disease, and *(b)* followed by conception within a reasonable time. In the group of 50 infected cows there were 150 terminations giving:

(a) a normal calf at term and *(b)* followed by prompt conception	36
Failure to meet requirements *(a)* and *(b)* at least once	31
Abortions	32
Aborted more than once	12
Aborted 3 times	3
Died of metritis	4
Crippled or poor	5

Twenty of the 36 normal records were distributed among 5 cows. Parturient disease was conspicuous in the group that failed to abort, the most frequent forms being retained placenta, metritis, and sterility. One individual delivered four calves, in four consecutive years, but three of them died of navel-ill. In thirteen cows abortion followed delivery of a calf at term. Retained placenta was recorded 33 times. In one cow retention was recorded five times and in another four. There were 100 calves and 50 abortions. Retained placenta occurred 22 times in cows that calved at term.

An accurate method of estimating the breeding efficiency of cows is to give a heifer calving not later than three years of age an "A," a cow conceiving in not more than six months after calving an "A," and a cow delivering a live calf at 265 days or more an "A." Failures are marked "B." With this code I have examined over 200 cows with more than 600 readings in herds where Bang's disease prevailed. Approximately 70 per cent "A" were found in the group reacting from 1:160 to 1:640, and 90 per cent "A" in the group from completely negative to 1:80. Nearly all that received an "A" ten times or more were in the

negative group. Nearly all receiving "B" two or more times were in the positive group. Cows making "B" three times, whether positive or negative, had a low inventory value as breeders. In general the score drops as the blood reaction rises, but a marked drop occurs in the group from 1:160 to 1:640. While there are examples of good breeding performance in positive individuals and groups, the average usually is low in any herd over a five-year period. The use of this code indicates the danger spots in the herd regardless of whether the cause is Bang's disease or some other influence.

A comparison of the breeding efficiency of high reacting cows with negatives and low reactors over the same period shows that among the positives the failures are three times those of the negatives and low reactors. One cannot deny the record, however, that groups of infected cows may reproduce in a fairly satisfactory manner. But in estimating the efficiency of such a group, one needs to include the members that have fallen by the wayside, the ones that have been sold, that were crippled, that died, that became sterile, that never bred, and to continue the observation at least five years in order to include the loss from a possible "storm." It is not difficult to record a satisfactory performance by a reacting cow, but it is impossible to predict which of a group of infected animals will reproduce normally. As a group they are often a source of loss; as measured by the value of their progeny and milk production they often fail to return the cost of their growth.

While a positive herd may reproduce in a satisfactory manner over a period of years, there is always the possibility of a disastrous epidemic.

Diagnosis.—In the case of an abortion the *placental lesions* may be so characteristic that a highly probable diagnosis is possible by means of an examination of the gross lesions. The leathery condition of the chorion is of special significance; this condition is infrequent in any other infection. Smears made from the margin of the necrotic areas on the chorion, or from the uterine exudate, practically always reveal the typical bacilli, both free and enclosed in cells. Additional confirmation is obtained by inoculation of a guinea pig with tissue from a cotyledon. Routine examinations of the placentae of a herd reveal information of great value concerning the presence of infection and of lesions in the uterus. Because of the frequency of abortions and of genital disease from various causes in all cows the question of suspected Bang's disease is constantly present. Routine guinea-pig inoculations of placental tissue may reveal *Br. abortus* before there are any gross lesions in the placenta, and before the blood reacts to the agglutination test. In this manner it may be recognized in recently infected herds before exposure becomes general. *Br. abortus* may also be found in the

fetus, especially in the stomach contents or the lungs. The usual diagnostic method is by means of a guinea-pig inoculation, or a culture of the stomach contents. The milk often carries the infection, but examination of the milk for infection in the cow is inferior to other methods.

The agglutination test of the blood serum has been universally adopted and is widely applied in the routine detection of infected cows. As a means of diagnosis for the purpose of separating the infected from the noninfected animals under a system of control it is highly satisfactory. But there are circumstances that restrict the value of the agglutination test. In recently infected herds, where the disease may be spreading rapidly, the bacillus may multiply in the uterus and even cause abortion before the blood serum will cause an agglutination. In such herds routine examination of placentae and inoculation of guinea pigs may greatly assist in the detection of infected animals. In some instances bacilli may be obtained from the placentae of cows that fail to show any change in the serum on subsequent tests, and it is probable that such cows are a source of exposure. A certain number of animals that become positive may finally show a reduced titer; this is chiefly observed in unbred animals, in vaccinated individuals, and in cows whose reactions are never over 1:200. When the reaction reaches 1:300 it usually goes higher and it rarely comes down. In recently infected herds the test should be conducted as often as once a month, and even then it may be impossible to prevent infection of the entire herd where the numbers are few. In negative herds, yearly tests are sufficient, except for individual animals that may abort or otherwise become suspicious.

As reported by the B.A.I.,[3] "The serum and whey agglutination tests and the whole-milk ring (ABR, Abortus Bang Ring) and plate tests were all highly efficient in classifying cattle as free of or infected with *Brucella abortus*." An extensive report on the use of this test in the field has been made by Roepke et al.[19] It is not recommended for use by individual herd owners.

In the *differential diagnosis* one needs to consider that abortions from other causes are frequent. In this connection it is of interest to quote from the writings of Smith and Little[20] in 1923: "Premature expulsion of the fetus in recent years has been quite universally ascribed to *Bacillus abortus* exclusively." In large negative herds the abortion rate is frequently from 5 to 10 per cent. Often these occur early in pregnancy, but they may occur at any time. An examination of the placenta, if obtainable, fails to show the typical *Br. abortus* in smears. Both the fetus and placenta should be examined by means of smears and guinea-pig inoculation in order to detect evidence of Bang infection as early

as possible. Usually the results are negative, but when a positive result is obtained immediate additional examinations need to be applied to the entire herd.

In general, abortions not due to the activity of *Br. abortus* react less seriously upon the cow than is the case in Bang's disease. Yet occasionally a negative cow will abort repeatedly and never deliver a live calf. Retained placenta is often interpreted as evidence of Bang's disease, but this condition is so frequent in all groups of cows that, taken alone, it has little significance. In genital disease due to other causes, usually there is either no evidence of contagion or it spreads slowly.

A survey of genital diseases encountered in our ambulatory clinic shows that of 253 aborting animals, 38.7 per cent were negative to *Br. abortus;* of 160 animals diagnosed as retained placenta after delivery of a calf, 80.6 per cent were negative; of 68 cows affected only with metritis, 80 per cent were negative; of 43 animals treated for dystocia, 83 per cent were negative.

In an infected herd, where a program against Bang's disease is in operation, any abortion should be followed by an examination of the placenta, or fetus, or both, including guinea-pig inoculations. In our series, in cases compiled from the ambulatory clinic, 50 per cent of the cows whose blood reacted at 1:80 at the time of abortion have also been positive in the milk, or the placenta, or the fetus. Cows that reacted at 1:40 at the time of abortion have been 16.6 per cent positive on other tests. Cows with entirely negative blood at the time of abortion have been 8.6 per cent positive on other tests. It is probable that abortions from cows showing a low blood titer at the time of abortion were in recently infected animals. While it is often stated that the blood of a reacting cow may develop a negative phase during advanced pregnancy and parturition, there is no evidence in support of this view. The blood of cows reacting in the lower range varies somewhat from time to time, but this variation is not influenced by parturition.

Control.—Two chief methods of control are employed: one is based on the diagnosis of infected animals by means of the agglutination test followed by separation of the infected from the noninfected group; the other depends on artificial immunization by means of inoculation with living *Br. abortus*.

Immunization.—Since the discovery of *Br. abortus* by Bang in 1897, the use of either live or dead cultures for purposes of immunization has been widely practiced and until recently there has been little to indicate that artificial immunization is possible. Yet this prophetic sentence appears in the report by Bang:[1a] "I am far from pretending that I have

solved the question of vaccination against abortion, but I think my experiments have made it probable that it will be possible in this way to get effective results. . . . Whether or not *vaccination* will in the future be the chief weapon against contagious abortion, time will show."

In 1924 Huddleson[21] issued a report on the use of a *nonvirulent living culture* of *Br. abortus* wherein he expressed the belief that an organism will not reacquire its disease-producing properties once they have been completely lost, and presented evidence that his avirulent culture did not cause premature expulsion of the fetus, and that it did stimulate the production of protective bodies.

In 1930 Buck[22] described an investigation to determine whether a lasting immunity could be engendered in bovines by their vaccination during calfhood. He concluded that a vaccine of *medium virulence (Strain 19)* was preferable, and that it produced an immunity that seemed to continue through the second gestation. Subsequently investigations by Buck[23] and associates have supported the view that calfhood vaccination with Strain 19 at an age of from 6 to 8 months produces a satisfactory immunity against natural exposure to Bang's disease under field as well as experimental conditions. It is available as a suspension of frozen live organisms dried and sealed under vacuum and packaged with sterile diluent to yield a single dose of 5 cc. administered subcutaneously, or 0.2 cc. administered intradermally.

In 1948 Huddleson[24] reported "that certain mucoid-growth phases of *Brucella suis* when prepared in the form of a vaccine (designated as Brucella M vaccine) and injected into normal guinea pigs, are capable of engendering in such animals a high degree of resistance against infection with all three species of *Brucella*." The advantages claimed for this vaccine are a low and transient postinjection titer, and protection of individuals in herds where abortion is active. It can be administered in all stages of pregnancy, and the agglutination disappears in less than 90 days.

Calfhood Vaccination.—In herds where abortion is common among first calf heifers, the loss from milk and progeny is enormous, and the prevention of such losses by calfhood vaccination commends its use in all commercial herds of this type. The value of vaccination in preventing the act of abortion in a first calf heifer cannot be overestimated. Whether or not she remains a spreader and reacts to the blood test may be of secondary importance since they do not retard milk production or the growth of the calf. The final objective is to acquire a negative vaccinated immune herd which may be officially accredited, and this requires disposal of vaccinated stock which remains positive at 30 months of age. It is commonly stated that calves vaccinated between 4

and 8 months of age usually return to negative within 4 to 8 months, or within 3 to 12 months.[25] "Under the revised procedure, animals vaccinated as calves and still showing blood serum reactions (titers) may remain as members of a certified herd beyond 30 months of age, if it is determined upon 30-day retest that the reaction is stabilized or receding at dilutions no higher than partial to 1 to 100."[3] In California, where chief importance in commercial herds attaches to the degree of immunity, heifers are vaccinated at from 7 to 12 months with expectation that they will be negative to the blood test at the end of the first parturition. In heifer calves from infected dams, 100 per cent were negative in dilutions as low as 1:25 four months after vaccination, while in a corresponding group of vaccinates from unexposed dams 61 per cent showed average titers of 1:200 (B.A.I. Rep. 1945). In a group of vaccinated calves raised in contact with infected cows, 22.22 per cent remained as persistent reactors (Birch et al.).[26] With increasing age, both the immunity and the blood reaction are prolonged by vaccination; 6 months is commonly reported as the most favorable age. Since it is doubtful that an animal vaccinated as early as 4 months will resist field exposure after two years, or that vaccinal immunity will resist a highly virulent infection, there is increasing interest in the protective value of revaccination, especially from owners whose negative vaccinated herds begin to break after a few years. In large herds that have been clean for years and in which calf vaccination is contemplated, Birch (1946)[27] advises segregating the calves before they are vaccinated and holding them apart from the main herd until they cease to react, this is done to avoid reactions which occasionally develop in the adults after the calves have been vaccinated.

In calves vaccinated intradermally the vaccinal blood reaction is said to disappear a little sooner than in calves vaccinated subcutaneously; the dose is 0.2 cc. of Strain 19 prepared for intradermal injection. There is general approval of calfhood vaccination because of the immunity established against abortions in first and second calf heifers, return of the blood reaction to negative, the possibility of developing a brucellosis-free accredited herd with some degree of immunity against reinfection, and an increasing preference by buyers for vaccinated stock. Milk ordinances have been adopted by several cities which require that after a specified date milk offered for sale must come from herds classified by State and Federal livestock sanitary officials as brucellosis-free. The United States Public Health Standard Ordinance and Code provides that within a period of three years after adoption all herds producing milk for pasteurization must be operating under Plan A (test and slaughter) or Plan B (test of herd, vaccination of calves, and

retention of reactors until they can be sold for slaughter without heavy loss to the owner).

Regardless of failure of experimental projects to support the value of Strain 19 in establishing negative animals at age 30 months,[26, 29] the success of field operations has led to an overwhelming campaign which has greatly restricted the ravages of contagious abortion in cattle, and may result in its complete eradication.

Adult Vaccination.—In December 1944 the Chief of the Federal Bureau, Dr. Miller, announced official approval of vaccination of negative adults in infected herds with a preference for calfhood vaccination. Apparently this approval was based on reports from California that, in brucellosis, vaccination is more successful in adults than in the young, as it is in other diseases. The chief objections to adult vaccination are the persistence of the blood reaction, and restriction of sales and interstate shipment. But in infected herds these conditions already exist and relief from losses caused by abortion is of chief importance; there is no immediate hope for either accreditation, or permits for interstate shipment. Under this plan in herds threatened or experiencing "abortion storms," all negative cattle not advanced more than four months in pregnancy are vaccinated, in order to immunize cattle not yet infected. When vaccination is followed by abortion, it is usually claimed that the cow was too far advanced in pregnancy or already infected at the time of the injection, or before there had been time for the development of immunity. Vaccination during an abortion storm may exert little immediate restraint, but it is reported that after the first year the number of abortions may be greatly reduced; this would occur without vaccination. With the exception of some of the states in the South, where Bang's disease is infrequent, both calfhood and adult vaccination are widely practiced, often in the same herd. Opinions vary widely concerning adult vaccination, but the extent of its current use should soon provide the experience needed to determine its value. Any measure that will reduce the number of abortions will meet with favor, even if it increases the number of animals which react to the blood test. As reported by Berman et al.[28] cows negative to serologic and bacteriologic tests for *Brucella* at parturition were revaccinated within 60 days; this resulted in a rapid rise in agglutination titer followed by a relatively rapid decline. A year after revaccination, fewer than 10 per cent of the animals were reactors. Adult cows vaccinated when nonpregnant return to a negative status more rapidly than animals vaccinated during pregnancy.

Test and Slaughter.—Segregation of animals reacting to the agglutination test has proved to be a successful means of eliminating Bang's

disease from many herds. This effort has been stimulated by requirements of public health officers that raw milk come from negative cows. On large farms, where separation of cows into different groups is not difficult, elimination of the infection may be accomplished. In small herds, where only a single stable is available, little can be accomplished when the disease is rapidly spreading, as in recently infected herds. The prospect varies widely according to the per cent of the herd found to be infected on the initial test, and the current activity of the disease. Whether the per cent of infection be high or low, success depends on ability to meet segregation requirements. If conditions favor contact with neighboring cattle at pasture, if removal of cows at calving time to a special stall is impossible, and if the owner is not fitted to comprehend and apply necessary precautions, efforts to secure or maintain a negative herd are largely lost.

During the progress of an active epidemic the blood test may fail completely. Cows that give negative blood today may abort tomorrow. If it is practical to take samples and to test every two weeks, there might be a possibility of keeping ahead of the infection. In general, blood testing after the initial test is repeated once a month, but where the disease is not active, once in six months may be sufficient. It is necessary, however, to apply at least two or three monthly initial tests. This shows whether blood near the dividing line is stationary or rising. Once a herd is known to be negative, once every six months to a year may be sufficient. The need for a retest depends on the environment of the herd. Many small herds are located where exposure rarely if ever occurs. Whether a herd be negative or positive, its performance is favored by continuous healthy growth in the calves and young stock, an abundance of exercise for males and females, the use of a disinfected box stall at parturition, an interval of at least three months after calving before breeding again, and of at least four months in the case of retained placenta or other evidence of metritis.

It is customary to segregate purchased cows for 60 days, and retest before admission to the herd, but this does not afford protection against nonreacting spreaders. Unless the herd of origin is accredited or known to be negative, replacement of cows by purchase may result in reinfection. While a nonreacting spreader usually becomes positive, it may remain negative for months, and an unknown and unrecognized few remain permanently negative.

When the tube method of conducting an agglutination test is used, dilutions are usually made at 1:25, 1:50, 1:100, and 1:200. Complete agglutination at 1:100 is regarded as positive; complete agglutination at not higher than 1:50 is regarded as negative. Under official testing and testing for purchase, where it is necessary to reach a decision on

only a single test, without knowledge of the herd of origin or the breeding record of the individual, rejections are often made when the blood serum agglutinates at 1:25. While the use of a fixed standard for interpretation of the agglutination test is usually necessary or desirable, in herds where one has access to the breeding records, and an opportunity to examine the placentae, it may be possible to retain as negative certain individuals whose blood reactions are within the lower positive range, as from 1:100 to 1:160. As Cotton[14] (1932) has pointed out, there is no definite titer at which infection in all animals disappears. In monthly tests the new positives are as likely to come from cows with a negative history, as from those that may have been positive at from 1:25 to partial at 1:100.

As a means of diagnosis, agglutination by the short or plate method has proved to be accurate, and in recently infected herds it may reveal infected cows before the blood of such animals is found to be positive by the tube method. Not infrequently there is a decided lack of agreement between the plate and the tube test on the same sample. And within 60 days after a sample of blood is reported as positive it may be negative ("false reaction"); this experience is met with in both positive and negative herds.

Because of variations in the activity of the disease, segregation methods that are a success in one herd may prove to be a failure in another. The most effective method is to remove all reacting animals from the farm. Where this is not immediately feasible they may be removed to another building on the same farm; even where it has not been possible to provide separate caretakers for the two groups, there is little evidence that infection is carried from the positives to the negatives by attendants. A less satisfactory method of separation is to place the positives by themselves in one part of the stable, and not turn them in yards or pastures occupied by nonreactors. In the case of an abortion in the positive group, this method is apt to prove a failure unless the caretaker apprehends the event and removes the case of impending abortion to an isolation stall outside the main stable.

There are two chief sources of failure either to establish a negative herd or to maintain one already negative. One is lack of control over outside contacts at pasture or through purchased additions, and the other is to provide sufficient separation between infected and noninfected animals in the stables, particularly at the time of parturition or abortion.

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BRUCELLOSIS IN SWINE

Definition.—Brucellosis in swine is an insidious disease in which the lesions and symptoms are often obscure or unobserved. As a clinical affection it is an occasional cause of abortion, orchitis, and arthritis. Gross lesions are confined to the genital organs, bones and joints. It is caused by *Brucella suis* (Traum). *Br. suis* is the cause of about 50 per cent of undulant fever in man, and it constitutes from 1 to 4 per cent of *Brucella* infection of cows' milk.

Etiology.—The prevalence of *Br. suis* infection in swine in the United States is not well known. Boak and Carpenter[1] obtained positive serological reactions in 0.19 per cent of 1,054 blood samples originating in New York State, and 1.89 per cent of 2,735 samples originating in the Middle West. Johnson and Huddleson[2] report that it is widely distributed in Michigan, is still more frequent in Iowa, and is more prevalent than our past knowledge has indicated. In Iowa about 2.5 per cent of all hogs react to the agglutination test—McNutt.[3] And Spink[4] writes that "in Minnesota and in Wisconsin most human cases are due to *Br. abortus*, but in Iowa, *Br. suis* is more commonly the cause of human disease." Swine brucellosis is most prevalent in the corn-belt states of the Middle West (Iowa, Illinois, Missouri) and in California. Naturally occurring *Br. abortus* and *Br. melitensis* have also been reported in swine,[5] consequently hogs must be regarded as a source of brucellosis

in both man and animals. And Dr. Jordan[6] has reported that human cases of undulant fever resulting from contact probably exceed in number those traceable to contaminated dairy products.

Because infected swine fail to abort or show other symptoms, the distribution of infection has not been recognized.

As a clinical disease *Brucella* infection in swine occurs only as an endemic in certain neighborhoods or on certain farms. Thus it has been reported by Howarth and Hayes[7] from the university farm at Davis, California, as abortion in swine from which two humans were infected; by James[8] as a cause of lameness and abortion; by Frei[9] in Switzerland as a serious affection of the male genitals of boars, abortions in sows, and undulant fever in humans; and by Thomsen[10] in Denmark as a disease of the male genitals and a cause of abortion in sows. The identification of *Br. melitensis* in swine in Iowa by Borts, McNutt, and Jordan,[11] in 1946, is highly significant in relation to human infection, and the growing opinion from public health officials that *Brucella* in food-producing animals must be eliminated. In the literature one finds statements concerning the disease and recovery from the disease where there has been no disease in the clinical sense, the animals under observation merely being carriers of *Br. suis*. Clinically, the male appears to suffer more often and more severely than the female, and aged hogs are more apt to be severely affected than the young. The fact that swine brucellosis is chiefly a disease of the male, and transmitted by the male, whereas bovine brucellosis in cattle is chiefly encountered in the female, makes a most conspicuous distinction between the two kinds of infection—Thomsen.[10]

Br. suis enters the body through the mouth, through contamination of the feed, water, or soil with semen, uterine discharges, urine, or droppings of infected animals, but probably the chief source of infection in the sow is mating with an infected boar.

Bacteriology.—Within the body *Br. suis* has its habitat in the lymph glands, spleen, liver, udder, kidney, testicles, accessory male sex organs and gravid uterus, and fetus. When the disease is active it is present in the blood stream, and it may remain localized in the bones and testicles for years. In disease of the male genitals it may be recovered from the seminal vesicles, epididymis, and prostate glands. Following abortion, Hayes and Traum[12] recovered it from the stomach contents of aborted pigs and from the chorion of aborting sows. After infection occurs *Brucella* may be spread for 6 months or more from the udder, vaginal discharges and urine. In pigs born of infected dams *Br. suis* has been found in the blood as long as 12 months after weaning (U.S.B.A.I. Rep., 1944). McCullough et al.[13] found *Brucella* in the submaxillary

lymph glands of 0.07 per cent (35 of 5,000 hogs) which "should in no wise be interpreted as representing the total incidence of infection in these animals. Ten of these were *Brucella abortus*, 11 *Br. melitensis*, and 14 *Br. suis*." This observation suggests that transmission of various species of *Brucella* to various species of animals is more frequent than indicated by experimental projects.

Outside the body it may survive in contaminated soil, water, or objects for approximately 3 months. In this respect it is like *Br. abortus*. It is destroyed by pasteurization. The viability of *Br. suis* in swine tissues survives a temperature of −10°F. for 30 days. Swine infection occurs chiefly through contact with infected swine. *Human infection* with *Br. suis* follows ingestion of infected cows' milk, or the handling or consumption of infected pork. As in *Brucella* infection in general, swine are often carriers of *Br. suis* infection when they are not themselves obviously affected. In the severe outbreaks described by Thomsen,[10] a stud boar, after serving an infected sow, transmitted the disease to 19 of 33 herds from each of which he had served a number of sows. As described by Frei[9] the disease was introduced by a diseased sow, which communicated the infection to one of three boars, and he served as a focus from which disaster spread.

Morbid Anatomy.—In nonclinical cases of reacting swine, Johnson and Huddleson[2] reported that gross changes in order of occurrence were enlargement of the spleen, hemorrhagic areas over the gastric surface of the spleen, congestion and swelling of the skeletal lymph nodes, infarcts and parenchymatous degeneration of the kidneys, suppurative metritis and mild enteritis. The following postmortem changes have been reported by Thomsen[10]: Infected boars show purulent or necrotic inflammation, sometimes calcification, of the genitals. These changes are found in the testis and seminal vesicle and most often in the epididymis; atrophy and hypertrophy of the testicles are common. In the sow the uterus is the chief site of obvious pathological changes, and these are present in both pregnant and nonpregnant animals. The uterine mucous membrane often shows numerous whitish yellow nodules from 2 to 4 mm. in diamenter, and there may also be a thickening of the uterine wall. This condition of the uterus has been designated as *miliary brucellosis* of the uterus in the sow. The placenta shows congestion, hemorrhage and edema, as in the bovine. The aborted pig fetus has the pathological and bacteriological characteristics of the bovine abort. In organs other than the genitals obvious pathological changes are infrequent. Among them Thomsen[10] mentions abscess of the spleen, the chest wall and the extremities, inflammation of the joints and tendon sheaths of the limbs, and emaciation.

Symptoms.—The chief symptoms result from diseased genitals in the boar, and abortion and sterility in the sow. In the boar there may be loss of sexual desire, swelling of one or both testes, and in chronic cases atrophy of the testes. Often the testicles are normal to inspection and palpation. In an acute attack from arthritis or orchitis the boar may be stiff or lame, show inappetence, and lose weight; there may be an enlargement of the joints. The incidence of abortion varies widely; it is less frequent than in the corresponding disease in bovines, and may be absent. McNutt[3] writes that the majority of abortions in swine in Iowa are not caused by *Brucella*. The individual fetus is expelled about the 72nd day without labor pains, and enveloped in the membranes—Thomsen. Early and unrecognized abortions are frequent. Failure to breed may be due to sterility in either the boar or the sow.

The blood agglutination test is of less value than in bovine brucellosis. Many pigs known to be harboring the causative organism are negative to the agglutination test, and similar cases have been found in adult infected sows. Also, the agglutination titer in infected swine tends to diminish much more quickly than in cattle. It is not a dependable basis for the disposal of individual animals but is of value in determining whether or not infection exists in the herd. Titers of 1:25 to 1:100 in an infected herd would indicate the presence of infection (U.S.B.A.I. Rep., 1944).

Control is based on disposal of the entire breeding unit, which may be replaced by purchase. It may also be replaced by removal of the pigs of an infected herd to clean premises at the time of weaning and test frequently for evidence of infection; there should be three negative tests before the herd is considered negative. Holm et al.[14] supplemented this method with the retention of breeding stock until it had become negative, approximately 6 months for sows and 9 months for boars; these animals and their progeny remained negative when breeding was resumed. This suggested the possibility of an immune response from *Br. abortus* vaccine, and 261 gilts and boar pigs were vaccinated with Strain 19. The post-vaccinal reaction varied from 1:25 at weaning time, 60 days, to 1:125 at 100 days and over and dropped to negative in about 2 months. In both experimental and limited field observations, the results of vaccination were apparently beneficial. In experimental vaccinated gilts 88.8 per cent farrowed normal litters against 22.2 per cent in the controls.

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CHRONIC MASTITIS

(Garget; Agalactiosis)

Definition.—Chronic mastitis is a progressive inflammation of the udder characterized by frequent or occasional acute activity. Either constantly or at intervals the milk contains flakes or clots, shows increased alkalinity, reacts to various biological tests, and reveals streptococci or staphylococci on blood agar plates. At all times fibrosis may be found on physical examination of the udder. Pathologically there is an atrophy of the glandular tissue, fibrosis, and in some instances suppurative infiltration or abscess formation. It is commonly reported that the chief bacterial agent is *Streptococcus agalactiae*. Cultures of samples examined at the New York State Veterinary College show that this streptococcus was present in 57 per cent of the udder infections of 892 unselected cows, and in 24 per cent of 283 udder quarters infected following trauma (Ferguson[1] 1943). Recent observations suggest that in frequency and severity staphylocci predominate in many herds.

History.—In 1884 Nocard and Mollereau[2] reported observations of an infectious form of mastitis, from which they isolated a pure culture of streptococcus from the secretions and reproduced the disease by injecting the culture through the milk duct. As related by Bang[3] in a

lecture in 1888 on *The Cause of Mastitis,* "one of them was consulted in 1884 by a cow-keeping dairyman in Paris who was almost in despair because his stock had been infected for six years with a terrible degree of mastitis. . . . Within 3 to 4 weeks after a cow was stabled there appeared a hard, usually painless, area in the lower part of one of the glands. The authors described it as an induration. It took months before it occupied one third to one half of the gland. . . . The milk finally became watery and yellowish." Nocard and Mollereau believed the milker transmitted the infection from one animal to another, since he did not wash his hands when going from one cow to another; before milking he wet the teat several times with milk from the bucket, and the film of milk left on the end of the teat might be the starting point for infection of the udder. In 1885 Kitt[4] reported similar observations on the bacteriology of mastitis. In mastitis milk he found the bacteria to be all of one definite form; these he was able to isolate and cultivate in pure culture; he reproduced the disease by injecting them into the teat canal through the teat duct, and he observed that normal milk within the udder was not infected. It is of special interest that the research workers who made these discoveries also observed the presence of corresponding changes in the udder and milk; they recognized the insidious onset, the beginning fibrosis, the early normal appearance of the milk, and the fact that it was a chronic rather than an acute disease. As related by Bang, "Nocard and Mollereau succeeded in overcoming the disease they were dealing with as soon as they insisted that the sick cows should be milked last and the sound glands in the sick cows milked before the affected ones, that the secretion of the diseased should be collected separately and used for swine feed, and that the milkmaid should wash her hands as well as the teats of the cow with a phenol solution (3 per cent) before each milking. In the cases observed by me the spread of the disease was indeed checked after such measures were established."

It is of interest to note that the chronic symptoms recognized fifty years ago by these veterinarians are commonly referred to by modern research workers as "subclinical" or "latent." Clinical differentiation of the "sound glands" from the "infected ones" for purposes of segregation and control, as successfully practiced by Nocard, Bang, and others, is now commonly regarded as impossible. The opinion of any layman that an udder is "apparently normal" is often accepted as final.

According to the kind of infection, the symptoms, and the tissue changes, various forms of chronic mastitis have been described; but when one observes a number of cases through successive lactation periods nearly all prove to be alike. The chief variations are in degree rather than character. Mastitis is constantly present in dairy herds

throughout the world. It is generally accepted as inevitable in the dairy industry and few efforts are made to bring it under control.

Etiology.—The causes of mastitis fall under two main groups: *(a)* the badly infected cow, and *(b)* insanitary stable and milking hygiene with special reference to the protection of the udder.

Fig. 99.—Chronic mastitis in advanced form. The white marbling represents extensive fibrosis. Proliferations are extensive around the teat cistern at the base of the teat. Note proliferations on the wall of the teat canal, and connective-tissue thickening of the lining of the teat duct.

(*a*) The Infected Cow.—When several cows with *diseased udders* are stanchioned among normal animals, the hands of the milker carry infection daily from the diseased to the normal udders. Apparently this constant application of infected milk to the ends of the teats finally leads to the entrance of bacteria through the teat canal, and from here

they invade the mammary glands. Reports of experimental transmission by means of natural methods of exposure have been almost entirely negative. In such experiments, a person first milks a badly diseased udder and directly thereafter a normal udder, keeping the hands moistened with the abnormal milk and leaving such milk on the teats of the normal cow. We have transmitted mastitis experimentally in this manner and in each case the first signs appeared after about three months in the form of fibrosis in the udder, pinpoint flakes in the milk, and a few bacteria on blood agar plates. At this early period the bromthymol-blue test was negative. While this evidence is not conclusive, it harmonizes with field experience. Seelemann[5] has described transmission experiments in 18 animals in which he tried to duplicate natural infection; only one was successful. The cow developed mastitis after a month's exposure on a bedding of straw sprinkled daily with infected milk. Since most of his experiments were closed as negative at the end of two to four weeks the time may have been too short. Bendixen[6] obtained experimental transmission by bringing infected milk in contact with a slight incised wound in the end of the teat, and Klimmer[7] has reported experimental transmission by causing the udder to lie overnight on material saturated with *Str. agalactiae*.

Natural infection is readily observed under usual stable conditions when a cow with a normal udder is placed in the midst of a group of cows whose udders are badly diseased. In from two to three months a beginning fibrosis may be recognized on clinical examination and in the course of a year the udder lesions may be distinct or marked. A further application of this principle is seen in herds with 50 per cent or more of badly diseased udders; when no precautions are taken against transmission, many of the remaining normal cows become diseased within six months to a year. And eventually practically every individual becomes infected, a suggestion that either natural or artificial immunity is improbable. The fact that a group of mastitis-free cows can be maintained in a healthy condition over long periods, if segregated from badly diseased individuals, is additional proof that infection is carried by the milker.

One should not disclaim knowledge of the manner of spread of mastitis merely because transmission experiments have been negative. In the usual stable environment a cow is exposed more or less constantly through various lactation periods to a number of infected animals and to numerous predisposing causes. These influences are not readily duplicated in an experimental project containing only a few animals and limited to a few weeks of time.

Sucking among calves and heifers has long been considered as a cause

of infection in first-calf heifers, and mastitis in the first lactation is of common occurrence in badly infected herds. Experiments conducted by Schalm[8] have definitely proved that *Str. agalactiae* may be transferred to the udders of calves by sucking among penmates fed on infected milk and that the streptococci may persist there until the first parturition and then be shed in the milk.

It is difficult to avoid sucking among calves that are pail-fed unless they are kept in individual pens. It may be partially prevented by tieing

Figure 100.

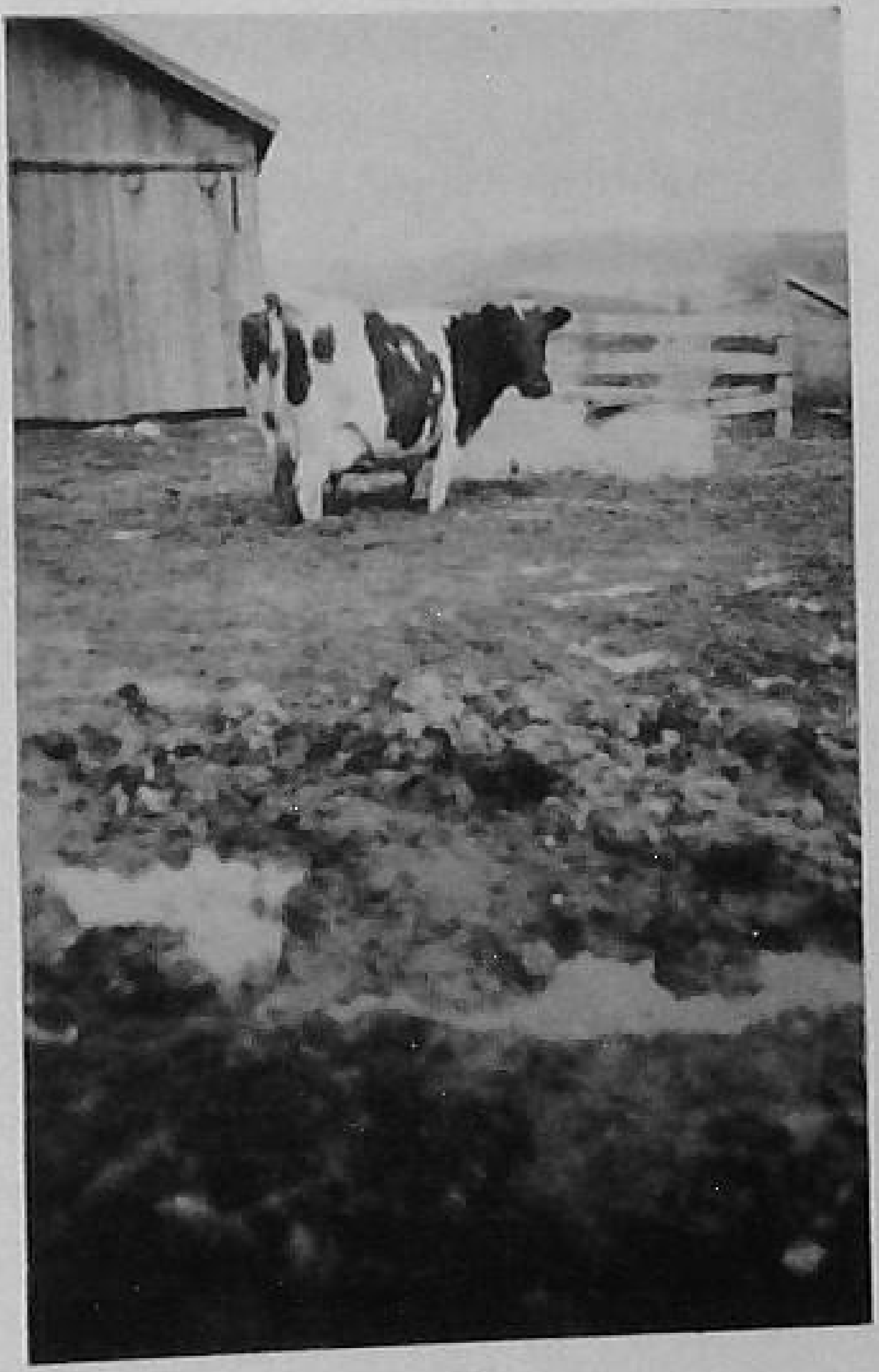

Figure 101.

Fig. 100.—A high door sill; one of the causes of mastitis.
Fig. 101.—This muddy yard caused a heavy loss from mastitis.
(Courtesy of S. D. Johnson.)

the calves in stanchions for one half hour after feeding. And calves raised on nurse cows do not suck each other. While it has been generally believed that the feeding of mastitis milk to heifer calves might result in infection of the udder, experimental feeding of such milk has failed to support this view.

Purchased Additions.—Often mastitis is widely prevalent in herds maintained by purchased additions. As high as 50 per cent or more of a consignment purchased dry may prove to be infected during the next lactation period. This may be explained in two ways: first, inflam-

matory changes are not easily recognized when a cow is "springing" or dry unless gross indurations are left in the quarter; and secondly experienced dairymen know that after an attack of mastitis the disease is apt to be permanent or recurrent, even though the acute symptoms may disappear, and it is from this group that cows are sold.

(*b*) Insanitary stable and milking hygiene and injuries may either initiate mastitis or accelerate its spread.

Injuries to the udder may be caused by faulty stable construction, hooks, or by sucking among heifers. Confinement in stalls that are too short, too narrow, without partitions, or slippery is a frequent cause of injuries. It is a common practice to tie both large and small cows in a stall-bed of only one length, and to stanchion Holsteins in stalls constructed for Jerseys. Often the stall is both too narrow and too short. These conditions lead to mechanical injury and to infection of the floor.

Fig. 102. *Left:* Stall beds too narrow. *Right:* Width of stall bed sufficient. Stall bed short for large cows. Freedom of movement allows udder to hang over edge of platform, or even reach the gutter.

Severe injury is often caused by being hooked, or bunted, or trod by other cows. Even when every precaution has been taken to prevent injury, such accidents are frequent. Injury to udders that drag over a high door sill at the stable entrance have been the cause of what appeared to be an epidemic of mastitis in herds whose milk failed to yield significant infection. A similar condition may be caused by milking with too high a vacuum or by leaving the milking cups on too long, where machines are used. In herds under routine inspection by veterinarians it is estimated that 85 per cent of the new cases which develop after removal of advanced cases of mastitis are due to injuries of the teats or udder. Even where the infection is high a large percentage of the new cases begin with an injury, and in such herds injuries usually result in mastitis. An injury that does not involve the teat meatus may cause

symptoms of mastitis, and the milk may be abnormal yet fail to show infection on laboratory examination.

Bedding.—Under usual conditions mastitis is invariably more prevalent when the cows have insufficient bedding; this exposes the udder to filth, moisture, and cold.

Milking.—It is a frequent observation that, as commonly used, milking machines spread infection. When improperly cleansed and disinfected a milking machine harbors infection constantly, and in herds where it is used, infected cows are more apt to be unrecognized than when milked by hand. Mechanical injury leading to mastitis may be caused by high vaccum of the milking machine, above 12 to 15 inches of mercury. In one herd a vacuum of 18 inches resulted in distinct clinical mastitis with "gargety" milk in a high percentage of the cows. The degree of infection was low and the condition of the udders improved rapidly after the pressure was reduced. Incomplete or irregular milking is harmful, especially in cows that already have some mastitis. A milker may fail to remove thoroughly the secretion from one particular quarter, as shown by the presence of fibrosis in the same quarter of each cow in his milking assignment. A cow that produces heavily may be a "slow milker," and after making a high record may develop udder trouble through incomplete milking. Mastitis often results from stenosis of a teat or cistern that requires care and patience in the complete removal of the milk. When udders require special care in milking, a change in milkers may be followed by an acute attack.

The following milking conditions favor the development of mastitis: *Rough manipulation* of the teat by pressing against it with the knuckles, or stripping it with the thumb and forefinger. *Wet milking* is so flagrant an offense against the quality of the milk and the udder of the cow that it should ban the milker from the stable. *Milking on the floor* is practiced in two ways: either as a means of discarding the milk from a badly diseased quarter, or because the milker is careless in directing the milk into the pail. *Failure to milk affected quarters* may result in a serious attack that might have been prevented; the apparently normal milk may be milked regularly and the milk saved, while the purulent secretion of the affected quarter is allowed to remain in the udder. In acute mastitis this practice is dangerous to both the cow and the consumer. *Milking tubes and dilators* in the hands of the average milker usually introduce an infection or aggravate one already present. *Milking with calves* is a common practice for the purpose of keeping the udder milked out in acute mastitis, when two or three calves are turned in with the cow. It is also employed as a "natural" method of raising calves. While it has been observed that a normal udder may develop

indurations when suckled by calves, this condition may be avoided by supplying a calf for each 10 pounds of milk production per day. Where nurse cows are used in this manner for raising calves, little milk accumulates in the udder at any time, infection if present is diminished, and in mastitis there may be marked improvement at the next lactation. Furthermore, calves raised on nurse cows rarely suck each other either before or after weaning. Infection sometimes occurs when the udder of a calf has been sucked by its mate after drinking milk; usually this infection is first recognized as a badly abscessed quarter when the heifer approaches her first parturition. *Washing the udder* and leaving it wet, in a cold stable, or where it may be exposed to cold drafts, predisposes to mastitis. And the repeated use of a cloth, without sterilizing, to wipe many udders exposes to the possibility of carrying infection from cow to cow.

Lactation Period.—The beginning and the end of lactation place a heavy strain upon the udder, and at these times a slumbering infection is apt to become active. Through this period the udder needs special care, a requirement that often is neglected in the routine milking practice of large dairy herds. When drying off, substitute hay for grain. If the milk-flow is especially persistent, the drinking water may be reduced to one pailful daily. A few abruptly discontinue milking; but the common practice is to milk once a day, once every second day, and so on until the flow has ceased. Whatever milk the cow gives should be entirely removed at each milking, and after the milking has been discontinued the udder should be watched for a time to recognize any return to lactation. When mastitis is distinct or marked, it may be impossible to dry off the udder without causing acute activity; in such cases it is better to continue milking and not attempt to give it a period of rest. Directly after freshening, injury may result from insufficient removal of the milk. When milking is left to the calf and the animal proves to be too weak to suckle normally, excessive engorgement may cause a severe acute inflammation. Similar injury is observed when the milk is allowed to remain in the udders of fresh cows on exhibition. Severe damage may result from the presence of pus in a quarter during the dry period, when the inflammation and the pus gradually increase. This may be prevented by the examination of dry udders and their secretions several days after the milking has been entirely discontinued; when pus is found, milking of the affected quarter should be resumed. In herds where monthly examinations of the udders are made, the dry cows should be included.

Size of the Herd.—Mastitis tends to be more prevalent in large herds because of less care in milking and less individual attention.

High Protein Feed.—It is commonly believed that a high protein diet may cause mastitis. There is no doubt that such a diet may aggravate a mastitis that already exists, but there is doubt that heavy feeding exerts any ill-effect upon the normal udder. But one needs to consider that in practically every herd there are some individuals affected with low-grade mastitis which may become more active when

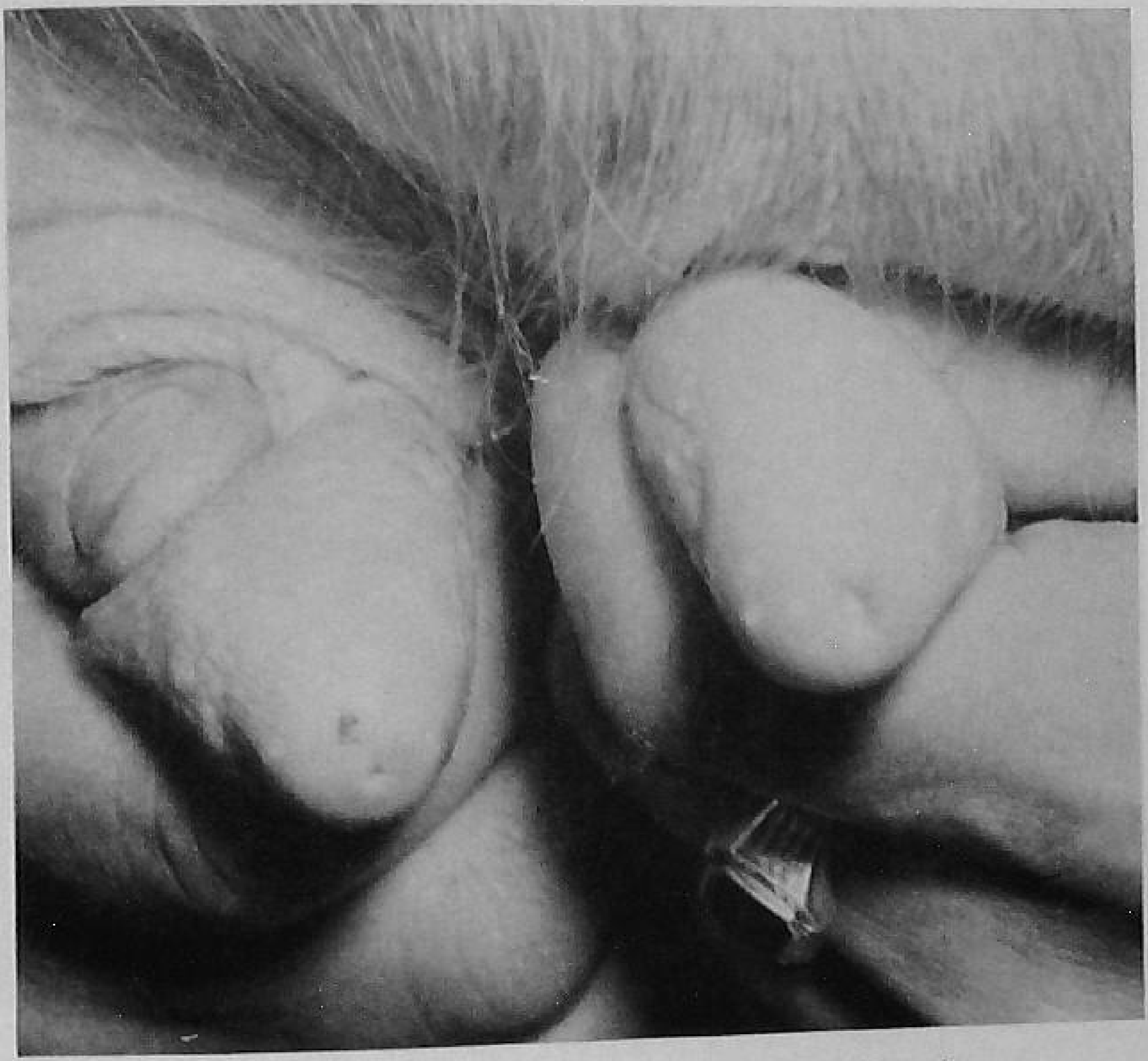

Fig. 103.—Normal teat meatus, left; slight dilatation (patent teat meatus), right.

a heavy grain ration is only moderately high in protein. The results of such feeding are readily observed when a group of cows affected with low-grade mastitis are fed a 12 per cent grain ration; the quality of the milk improves as shown by the decrease of flakes and clots on the strip-cup.

Associated Diseases.—According to the theories of Pröscholdt[9] and others, abortion infection seems to mobilize the "slumbering streptococci," and recently a few writers have stated or inferred that the direct effect of Bang's bacillus on the udder largely explains the diminished flow of milk associated with this disease. Clinicians recognize a relation between severe acute affections of the uterus and inflamma-

tion of the udder. Sometimes mastitis is associated with septic metritis, but I have found no record of a bacteriological study of this condition. In general there is no relation between the control of abortion and the control of mastitis. While the udder may be the habitat of Bang's bacillus, there is no evidence that this infection is either a direct or a predisposing cause of mastitis. Epidemics of cowpox may result in severe mastitis if the pox eruption spreads to the ends of the teats.

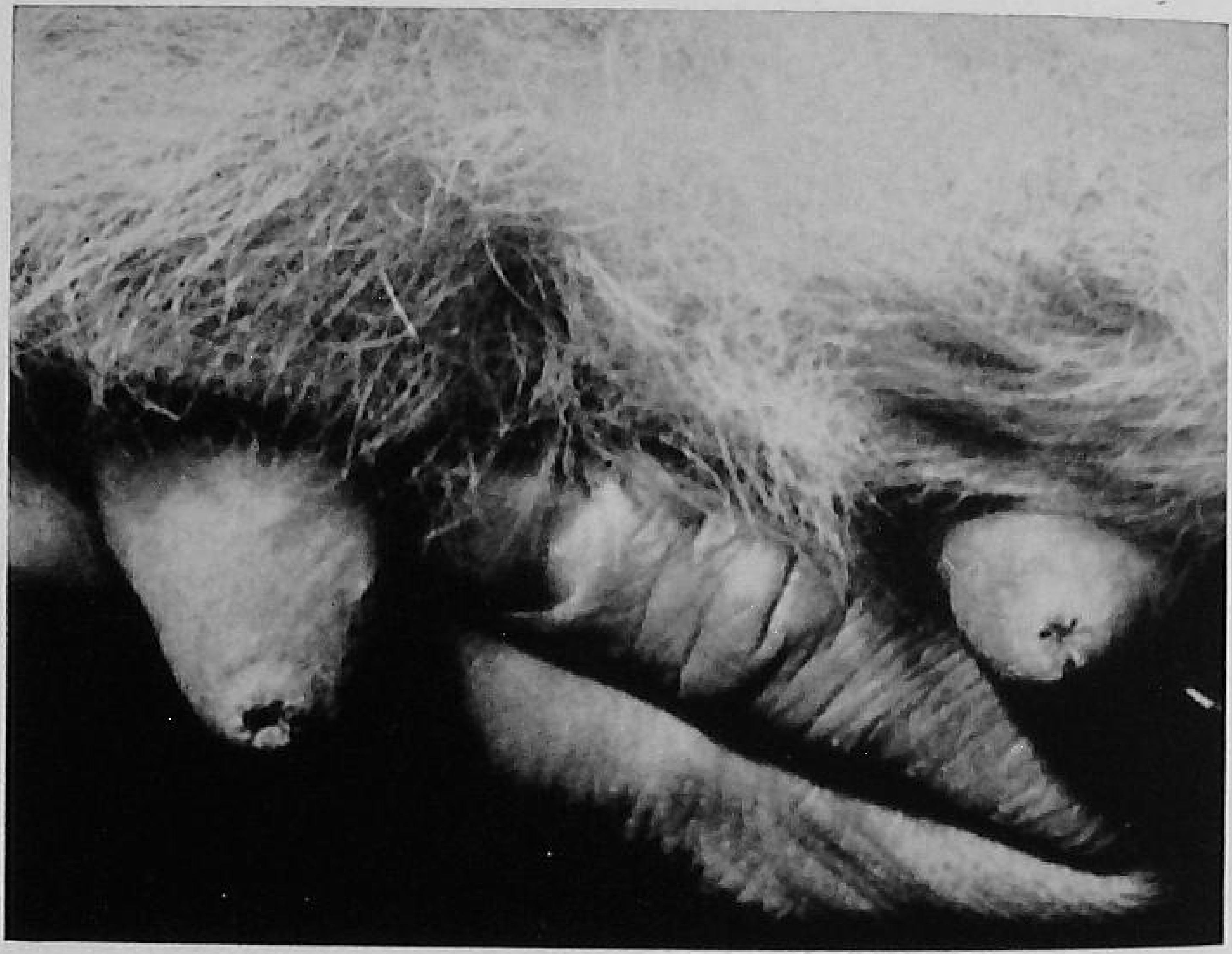

Fig. 204.—Teat erosions: meatus prominent and open, left; meatus prominent, closed, and proliferative, right. (Courtesy of S. D. Johnson.)

Age.—It is a common observation that the disease is more prevalent among the older cows. Few aged cows that have been heavy producers are entirely free from fibroses. Where old and young are together in a badly infected herd, the rate is lower in the younger group. Age alone, however, will not bring mastitis. In herds under a control program the aged as well as the young may have healthy udders; where mastitis is prevalent, each year of exposure reduces the chance of escape.

Patent Teat Meatus.—A flaccid open teat meatus may be either congenital, or acquired by an injury to the end of the teat. This term applies to a more flaccid condition than found in "easy milkers," though there is no distinct line between the two. It has been observed that infection less readily enters when the teat meatus is tight. If the teat

meatus is so relaxed that milk starts whenever the teat is handled lightly, infection is almost certain to enter the gland and cause chronic mastitis. The nature of the infection in such cases is variable; often it is a staphylococcus and it may vary in different samples of milk.

Teat Erosion.—This term refers to a variety of lesions at the end of the teat, usually involving the meatus, and caused by milking machines. The most frequent lesion is a prominent meatus, giving the appearance of an eversion of the meatus, and presenting a more or less prominent white ring around the opening of the teat. Associated with a prominent meatus one often finds wart-like fine or coarse projections, and these may be more readily felt than seen; in extreme cases they cause stenosis of the teat meatus and proliferate and crack open across the end of the teat. In nearly all there is redness of the teat end which is more prominent as soon as the teat is handled. Less frequently the teat meatus is flaccid and open (patent); occasionally it is entirely closed, and in extreme cases the teat canal and the udder are involved. These lesions develop within a few days after operation with the milking machine, and usually disappear when the cow is dry. They are most frequent where the vacuum is as high as 15 inches, but may appear to a lesser degree and less frequently with a low vacuum. In certain herds there may be no evidence of teat erosions even with a high vacuum. The condition is aggravated by leaving the teat cups on too long. Aside from cows that are affected with such severe lesions that they have to be milked by hand, and possibly with a milking tube, there is no definite evidence of the relation of teat erosions to mastitis. There is a suggestion, however, that these changes are often associated with bromthymol-blue reactions, and with various degrees of chronic staphylococcus mastitis. The manner in which a teat cup may erode the end of the teat has been described by Espe.[10] The area and degree of compression of the teat cup may be recognized by inserting the thumb into a cup while the machine is in operation. There is need for research on the relation of milking machine operations to the health of the udder, as well as the effect of different types of machines.

Bacteriology.—*Streptococcus agalactiae (mastitidis)* Lancefield Group B, is the name now applied to the streptococcus isolated in pure culture and described by Nocard[2] and others in the 1880's. Sherman[11] writes that its precise identity may well be dated from the work of Ayers and his co-workers, 1918-1922. As reported by Sherman,[11] (p. 27): "these investigators correlated the production of a low pH in glucose broth, limited and variable hemolytic power, and the ability to hydrolyze sodium hippurate in the differentiation of the mastitis streptococcus

from hemolytic streptococci of human infections . . . the sodium hippurate reaction has remained, for its specific purpose, one of the outstanding differential tests in bacteriology . . . another character which appears to make the mastitis organism somewhat unique among the hemolytic streptococci is its apparent total inability to attack esculin." According to Schalm,[29] "This organism does not invade tissue; its effect upon secretory cells is, therefore, through an irritant formed in the milk." And on the contribution of Lancefield, Sherman[11] writes, (p. 13): "The most important contribution to methods for the classification of streptococci is the serological technique of Lancefield (1933) which divides the hemolytic streptococci into groups by means of a precipitin reaction . . . the scientific and practical results achieved rank the single-handed Lancefield work with few peers in the annals of modern bacteriology."

The term *"hemolytic streptococcus,"* as used in relation to mastitis is often confusing. There is a beta hemolytic streptococcus (*Str. agalactiae*), Lancefield's Group B, which only causes mastitis in cows, and a beta hemolytic streptococcus (*Str. pyogenes*), Lancefield's Group A, which causes septic sore throat in man. Mastitis in bovines is also infrequently caused by transmission of *Str. pyogenes* from man to the cow's udder, when the milk is capable of causing a milk-borne epidemic of septic sore throat. But apparently this infection is incapable of passing from animal to animal. There also may be a direct contamination of milk with *Str. pyogenes* when the handler thereof is a carrier.

According to the appearance of the growth on blood agar plates, three distinct strains of *Str. agalactiae* are recognized: nonhemolytic colonies, narrow-zone hemolytic colonies (Fig. 105), and broad-zone hemolytic colonies (Fig. 106)[26, p. 169]. In an infected herd one or the other of these strains often predominates, and occasionally only a single strain is found in the milk samples of a herd. Those giving either nonhemolytic or green colonies are less frequent and the narrow-zone colonies are the ones most commonly present. This prevalence of a particular strain in the herd is proof of its spread by contact, of its dependence on the udder for permanent existence in the herd, and of its introduction by a cow with a diseased udder. Between the different strains of *Str. agalactiae,* there appears to be a low virulence in the nonhemolytic type.

Str. agalactiae has been recognized as the chief infection in chronic mastitis,[25] and it is still believed by many to be the chief type of infection in herds where no special control has been applied. Its historical predominance has almost conferred upon udders the status of a specific infectious disease. But where udders have been treated with in-

fusions of antibiotics or other appropriate disinfectants the agalactiae appear to be least resistant to their action and other forms, especially staphylococci, predominate. There have been numerous discussions on whether *Str. agalactiae* might inhabit the udder without causing mastitis, or whether in such alleged habitat the pathological condition was present and unrecognized. There have been instances in which laboratory examination of milk samples from herds have yielded agalactiae when no clinical mastitis was recognized on examination by experienced veterinarians. In the writer's experience, when the "limited and variable hemolytic power" of this species recovered from milk samples was determined to be nonhemolytic, or narrow-zone, or broad-zone hemolysis, there built up a suspicion that the nonhemolytic strains might be nonpathogenic. Until additional observations are available the question will remain unanswered.

The Habitat.—The habitat of *Str. agalactiae* is chiefly in the diseased udder and its numbers are in direct proportion to the extent of the lesions. Its presence in the milk of normal cows has often been reported, a view that was emphatically opposed by Kitt[4] as early as 1885, and by Seelemann[5] in 1932. None of the earlier authorities, who were trained in physical diagnosis and able to distinguish between a diseased and healthy udder, reported that the normal milk itself within the udder carried infection. In our experience in badly diseased herds, where a third to a half of the cows have been affected, *Str. agalactiae* has occasionally been found in the milk of an individual whose udder was normal on physical examination. Where infection is abundant it may sometimes be recovered during the period of incubation. But one cannot be certain that such individuals will develop mastitis, since spontaneous loss of infection is sometimes observed in cows under routine examination. Differences of opinion on this point are probably explained by the wide variations in the clinical examinations of the udders. Conclusions based on a milker's statement that an udder is normal, or on a superficial examination of the udder, or on an examination by one who lacks training in palpation of the udder are unreliable. When the lesions are advanced, mastitis streptococci are eliminated almost constantly in the milk. In certain individuals it may be intermittent, and occasionally milk from a badly diseased quarter fails to show streptococci on blood agar plates in examinations made over a period of weeks. Elimination may also vary in the same animal in different lactations.

Many attach important practical significance to the "subclinical" or the slightly infected udder as a source of infection and deny that the badly diseased udder is the chief source. According to this view diagnosis by clinical examination, as used by Nocard, and Bang, and others,

is inadequate; there must be a bacteriological examination. This widely held view is expressed by Edwards[12] as follows: "During extensive studies on this condition it has been realized that a large proportion of infected animals are affected in the latent stage and that one of the main problems in its control is the detection of these animals." This view is only a slight modification of the claim that the normal udder is the habitat of *Str. agalactiae*. The great majority of so-called "latent" cases of mastitis are accounted for by lack of examination of the udders and not by their "subclinical" condition.

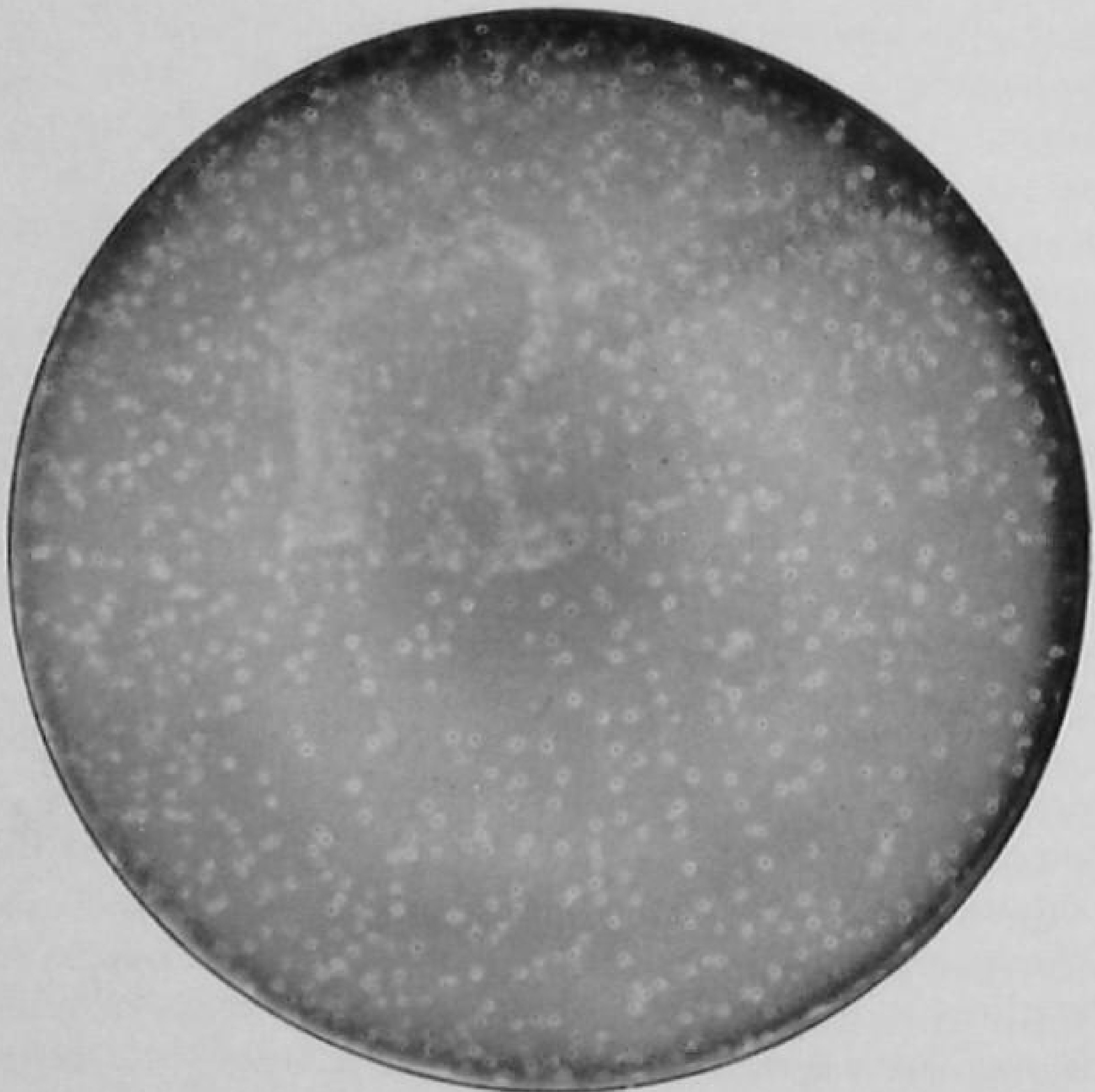

Fig. 105.—Narrow zone hemolysis of *Streptococcus agalactiae*.

The relation of *Str. agalactiae* to other organisms in the udder is variable. In certain herds under routine examination it has been observed that staphylococci were regularly recovered in large numbers from quarters that subsequently yielded only streptococci. In herds only moderately diseased, or under a system of mastitis control, the per cent of *Str. agalactiae* has been relatively low while staphylococci and other types of mastitis streptococci have been more frequent. Under the influence of a control program, agalactiae gradually disappear and an increasing percentage of subsequent infections are either staphylococci

or other types of streptococci—organisms that presumably are constantly present on the premises. In herds infected with *Str. agalactiae* the organism is capable of existence outside the udder, and where pus is milked on the floor the degree of stable infection must be high. Little is known of its capacity to survive outside the body, but apparently its existence there is not permanent. This view is supported by the observation that where the degree of infection with *Str. agalactiae* is light, mastitis caused by injuries usually yields some other type of infection. Where *Str. agalactiae* has been eliminated, as shown by quarter samples

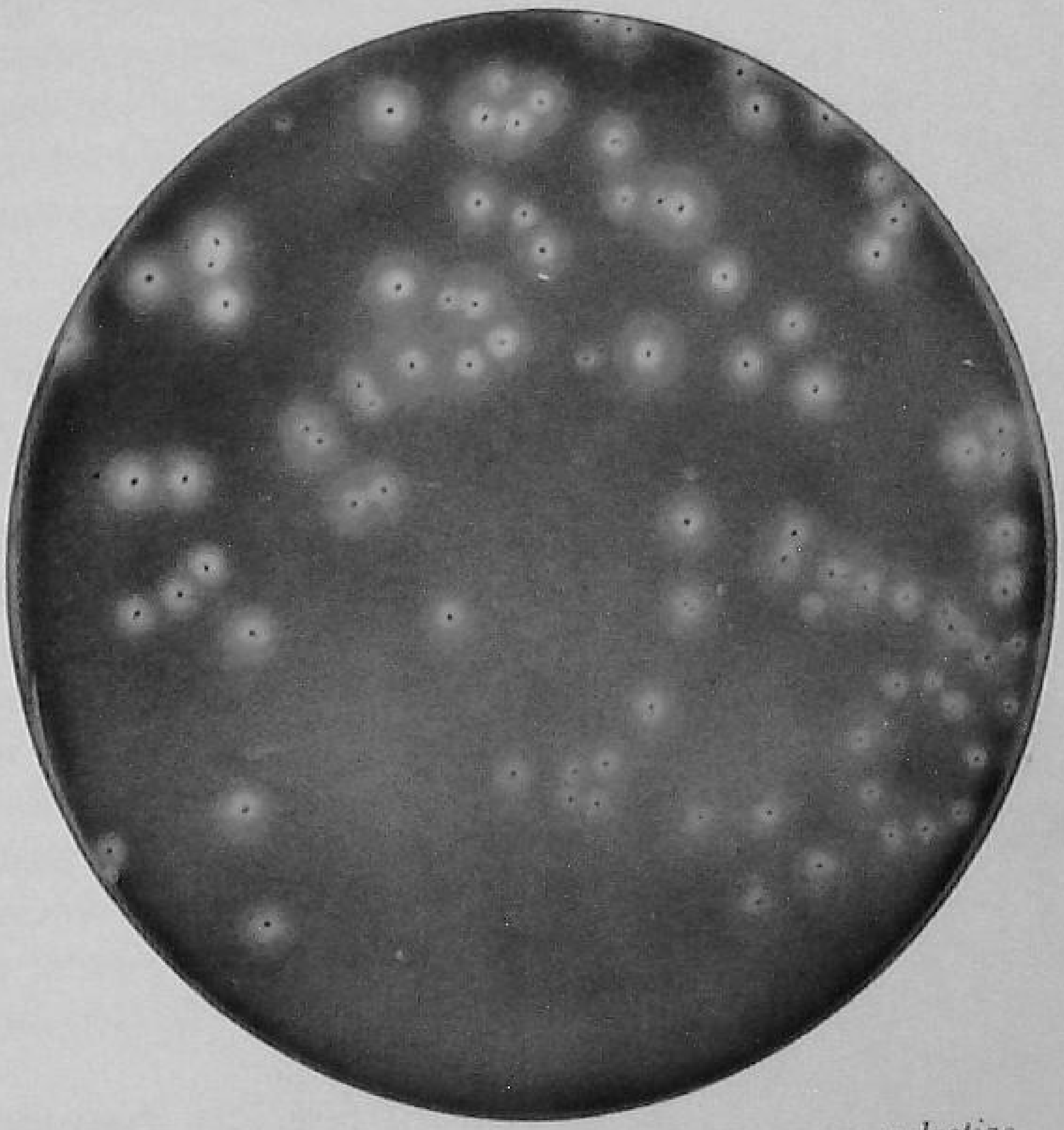

Fig. 106.—Broad zone hemolysis of *Streptococcus agalactiae*.

of milk, it is not found in injuries to the teats. The term streptococcic and staphylococcic mastitis are almost universally used, and it has been suggested by bacteriologists that the term mastitis be replaced by the name of the infective organism. Yet a survey of the lifetime yield of cows affected with mastitis discloses variations in the bacteria in different quarters of the udder in any single sample of milk, as well as variations in the same quarter taken in different lactations, or even in a single lactation. While there are exceptions in which some particular organism may persist uniformly, my collection of lifetime laboratory

reports of several hundred cows shows that variations are the rule, and that identification according to the yield of a milk sample usually is transient.

In market milk, mastitis streptococci may be found in practically all samples, either raw or pasteurized—Brown.[13] Sherman and Niven[14] examined a total of 313 samples of commercial milk, 68 raw and 245 pasteurized. "Narrow-zone hemolytic types in blood agar, the most typical form of *Streptococcus mastitidis,* were not considered. Only 8.5 per cent of the pasteurized samples contained hemolytic streptococci, whereas broad-zone hemolytic types were obtained from 18 per cent of the raw samples." None of these were human pathogens—Lancefield's Group A. The prevailing types of hemolytic streptococci in raw milk were *Str. agalactiae.* From these reports one may conclude that practically all milk contains hemolytic streptococci of a type not pathogenic to man.

Resistance.—Little has been reported on this subject. Seelemann[15] found that *Str. agalactiae* survived a temperature of 85°C. (150°F.); high resistance is explained by their enclosure in masses of leucocytes.

Str. dysgalactiae and *Str. uberis,* termed green streptococci because they show nonhemolytic green colonies on blood agar plates, are the most frequent types of infection following injuries to the teats: 38 per cent as compared with 24 per cent *Str. agalactiae,* according to compilations by Ferguson.[1] Infection with green streptococci has been frequent in outbreaks of mastitis following cowpox, and in mechanical injury from an improperly adjusted milking machine. When an unusual number of milk samples yield green streptococci one may suspect mechanical injury. Clinical evidence of such injury is shown by a thickening of the teat duct, an eversion of the teat meatus, and scabs or a red area suggestive of an injury, but regular in outline and located directly on the end of the teat.

The habitat of green streptococci (*Str. uberis* and *dysgalactiae*) organisms appears to be on the premises, where infection may follow injury or take place in a manner entirely unknown. There is no evidence that the mode of infection is by direct or indirect contact between animals, but such infection may possibly occur from a badly diseased udder. These organisms cause a type of mastitis that cannot be distinguished clinically from that due to *Str. agalactiae.* It is sometimes stated that they cause only a mild acute attack with a tendency to recover, and that the infection is transient. Such cases are observed, but the acute attack may occasionally leave the udder worthless. In our experience the majority have been chronic, as shown by routine physical and laboratory examinations. In some of the chronic cases

the infection disappears spontaneously and a gland that appeared to be badly damaged returns to normal. Occasionally, however, *Str. dysgalactiae* or *Str. uberis* is the only infection found in a hopelessly damaged No. 4 udder.

Staphylococci are a frequent cause of chronic inflammation of the udder. They may be hemolytic or nonhemolytic; they may occur alone or in association with streptococci, and they may be the chief infection in the herd. A few staphylococci on blood agar plates are of no significance, and often many staphylococci may persist for a time in a normal udder. But when a quarter yields many staphylococci continuously there is usually some degree of mastitis. Occasionally staphylococcus is the only infection found in a hopelessly damaged No. 4 udder. After an injury, as from being hooked, staphylococcus may be the only organism recovered from a quarter that is completely lost after two or three subsequent lactations. It is probable that the influence of staphylococci as a cause of chronic mastitis has not been fully recognized. In many cases, in examinations of milk covering the period from the time when the udder is normal until fibrosis is distinct, staphylococci are the only bacteria which appear on blood agar plates. It is improbable that injury is the only cause of infection. But there are wide variations in susceptibility to infection and it is not advisable to keep a cow with a badly diseased udder in the milking line, regardless of the kind of infection.

In a six-year summary of laboratory diagnosis of mastitis in Iowa, Packer[27] reported that in 15,693 samples of milk percentages of infection were:

Staph. aureus	72.60
Str. agalactiae	7.78
Str. dysgalactiae	4.4
Str. uberis	5.5
Escheria coli	4.25
Pseudomonas pyocyaneus	2.75

As described by Little and Plastridge,[26] nonhemolytic staphylococci may be regarded as nonpathogenic, and hemolytic staphylococci may result in a chronic mastitis which is usually less severe than that caused by *Str. agalactiae*. There is a prevalent opinion, however, that streptococci cause a less severe form of mastitis. This is expressed by Schalm et al.[29] who write that "Extensive use of penicillin and other antibiotics by dairymen has lessened the importance of streptococci in the mastitis complex. An etiologic shift now points to *Micrococcus pyogenes* (hemolytic staphylococci) as the primary cause of chronic mastitis since this pathogen is more refractory to the intramammary therapy and

readily dominates other flora in the absence of *Str. agalactiae.*" Unless the program to suppress agalactiae or any other infection is accompanied by improved stable and milking hygiene, which are the dominant factors, one or more infections will persist. This is suggested by the lengthening list of organisms under indictment since the extensive use of antibiotics. The type of infection is less important than the type of housing, the premises, and the method of milking. In different herds a bacteriological survey may reveal a predominance of either staphylococci or streptococci. A suggestion of the possibility has been revealed from data collected by Udall from about 100 cows over lifetime lactations in a large well-managed herd. There was an almost equal division between staph. and strep. with every possible combination and sequence. More than half had a record of carrying both, often in the same lactation or in different quarters of an individual milk sample. Infection came and went with or without treatment. Such an observation supports the view that some factor other than bacteria is dominant. This of course is self-evident when the end of the teat has been trod or attacked by cowpox or otherwise injured.

Pseudomonas pyocyaneus has appeared recently in udders that have been treated by the owner with infusions of penicillin, and Tucker[28] has reported finding this organism as a contaminant in empty vials and syringes in a stable where *Pseudomonas* mastitis followed such infusions. From California, Schalm[29] writes that mastitis associated with *Pseudomonas aeruginosa* and exotic organisms is being encountered with greater frequency and may be a consequence of both extensive use and abuse of antibiotics.

Escheria coli is the cause of an acute severe, often fatal mastitis (coliform) which occurs more often just before or shortly after parturition when the udder is large and distended. Another similar type is caused by *Corynebacterium pyogenes* which is rather infrequent in this country, but a cause of severe loss in Great Britain where it occurs both in lactating and nonlactating animals, even in unbred heifers, and is commonly termed "summer mastitis."

Microscopic Examination of Milk.—Microscopic examination for the detection of mastitis is made by various methods: direct smear of nonincubated milk (Breed smear); direct smear of incubated milk; and smear of centrifuged sediment. By these methods one examines individual quarter samples, composite samples from the quarters of each cow, or weigh-can samples of the mixed milk of the herd.

In a stained direct smear of *nonincubated* milk (Breed smear), one examines for streptococci, epithelial cells, and leucocytes. For detecting mastitis streptococci this method is less successful than cultures on

blood agar plates. When streptococci are found in nonincubated direct smears of samples of individual quarters it indicates extensive disease in the udder, and usually in the herd. As a rule mastitis streptococci are not seen in direct smears of nonincubated mastitis milk from individual quarters; even when the milk itself is obviously changed they may not be found. Exceptions are sometimes met with in badly infected herds; in a routine experience over a period of ten years we have found only two herds of this character. Minett, Stableforth and Edwards[16] found in a series of 223 culturally positive cases that diagnosis by microscopic examination of centrifuged milk sediment could be made in only 52.5 per cent.

Microscopic examination of smears from quarter samples aseptically drawn and *incubated* at 37°C. for from 12 to 24 hours is in common use as a method of diagnosing streptococcic mastitis. Since the laboratory requirements of this method are simple, it has wide application in practice. Contamination of samples is frequent, and in the interval between their collection and arrival at the laboratory growth of contaminants may render the milk unfit for examination; this is especially liable to occur when samples are sent through the mail without refrigeration. To suppress the growth of contaminants brillant green is placed in each sample tube: dissolve 0.1 Gm. brilliant green in 100 cc. distilled water, sterilize, and use 0.5 cc. for each 25 cc. of milk; this makes a 1:50,000 solution. Observations reported by Bryan[17] on samples held at room temperature for from five to seven days to simulate the time required for samples to reach the laboratory if sent through the mail, indicate that the most effective preservative was prepared by adding 0.2 Gm. brilliant green, 0.75 Gm. sodium azide, and 10 Gm. of dextrose to 200 cc. distilled water; this solution was autoclaved and 0.1 cc. added to each 5 cc. of milk. In routine examinations of milk samples that have been taken without regard to the condition of the milk or udder, there is an occasional report of the presence of *Str. agalactiae* or other streptococci when the milk fails to show any other evidence of infection, and the udder is physically sound: such reports may result from the use of either incubated smears or blood agar plates. A check sample on such reports is often negative, when one may assume the presence of a transient infection, or contamination. When the streptococci are numerous and the cell count is high, there can be no doubt concerning the diagnosis.

Of course it is impossible positively to identify *Str. agalactiae* or other mastitis streptococci in smears, but it is possible to identify them when milk from the incubated samples is cultured on blood agar plates and the colonies are run through the differential media.

Weigh-can samples of milk are always contaminated and they are always somewhat incubated. The presence of long-chain streptococci with or without many leucocytes in weigh-can samples is commonly accepted as proof of mastitis. One needs to consider that such samples are contaminated, that contaminants are the first to multiply under incubation, and that often they appear as long-chain streptococci indistinguishable in smears from mastitis streptococci. While smears of such samples may show abnormal constituents in the milk this may not imply that the udders of origin are abnormal. A herd must be badly diseased to show evidence of mastitis in smears of weigh-can samples, and when milk from badly diseased herds is properly cooled mastitis streptococci are not apt to be found in Breed smears prepared from such samples. Without any special study of the relation between streptococci and cells in smears of weigh-can samples, and the udders which provided the samples, there has developed a code whereby a diagnosis of mastitis in the herd is made from smears in the laboratory. The presence of streptococci in all market milk, and of some degree of mastitis in all dairy herds, makes it difficult to distinguish between the error and the accuracy of such decisions. From grossly diseased and mismanaged herds the milk may be sufficiently infected to show mastitis streptococci in Breed smears from weigh-can samples; usually the presence of streptococci in smears of weigh-can samples is due to improper care of the milk.

A high cell count may rarely be in the millions and transient from no apparent cause, and normally it is high in late lactation. With proper regard for the lactation period the cell count is useful in the determination of the presence and extent of inflammation of the udder. It is high when cows rapidly decrease in milk flow from a disease, such as winter dysentery. Often it is especially high in mild transient or constant staphylococcus infections. The degree of mastitis and the prognosis may be judged somewhat according to whether the count is in the two millions, the five millions or the ten millions. There are individual exceptions, however, so that no definite number alone can be accepted as a diagnosis of mastitis. According to Seelemann[5] the normal fluctuations in numbers of leucocytes are so great and the sensitiveness of the mammary glands so variable that the establishment of a fixed normal would only lead to error. He states that milk is a product of such variable composition that no fixed boundaries can be set. To determine accurately the significance of a high cell count, whether from individual or weigh-can samples, one needs to examine the udders of the suspected cows.

In a report by Johnson and Trudel[18] on the significance of leucocytes in milk, it was observed that "in cows whose udders are classed as Nos. 1 and 2 it is apparent that the number of cells in the milk increase during the latter stages of the lactation period. . . . In mastitis there is an increase in cells in the milk that corresponds to the degree of change found on the physical examinations of the udder, the changes found in the milk, and the bacteriological findings. In mastitis there appears to be an increase in leucocytes that precedes the appearance of streptococci or staphylococci in the milk." Our observations indicate that the cell count is of value in drawing the line between Nos. 2 and 3, when the increase persists they soon pass to No. 3. When the cell count reaches the millions per cc., and persists, it indicates infection, advanced mastitis, and pus in the udder.

For *direct microscopic examination* the sample should be taken aseptically from the foremilk without discarding the first 2 to 3 streams. First wipe the end of the teat with gauze or cotton moistened with alcohol and collect in a sterile tube. Smears may be made directly from the milk, or from centrifuged sediment. Various stains are suitable, but the Newman stain is widely used. It consists of methylene blue powder, 1 to 1.5 grams; 95 per cent ethyl alcohol, 54 cc.; technical tetrachlorethane (Eastman Kodak Co.), 40 cc.; glacial acetic acid, 6 cc. Add alcohol to the tetrachlorethane in a flask and heat to a temperature not to exceed 70°C. Add combined solution to powdered methylene blue. Shake vigorously until the dye is completely dissolved. Cool the solution and then add the glacial acetic acid very slowly. *Directions:* 1. Prepare milk smear. 2. When dry, immerse smear in solution and withdraw immediately and dry. 3. Wash in water. 4. Dry and observe. Streptococci in long or short chains indicate mastitis, especially when associated with leucocytes. In the past, classification of mastitis streptococci has been based on variations in the arrangement of streptococci in smears, such as long-chain streptococci, short-chain streptococci, diplococci, etc. According to the more recent work of Seelemann,[5] Rosell,[19] and others these variations do not indicate a corresponding difference in the streptococci or the mastitis produced by them.

Symptoms.—While mastitis is widely prevalent, knowledge of its symptoms and course is often lacking, especially in this country. Reference to a case as recovered when the acute symptoms recede is almost universal, even among veterinarians. To a dairy inspector, recovery has been accomplished when the gross appearance of the milk is normal. The specifications of the laboratory are met when apparently normal milk is free from mastitis streptococci. In mastitis, as in most other

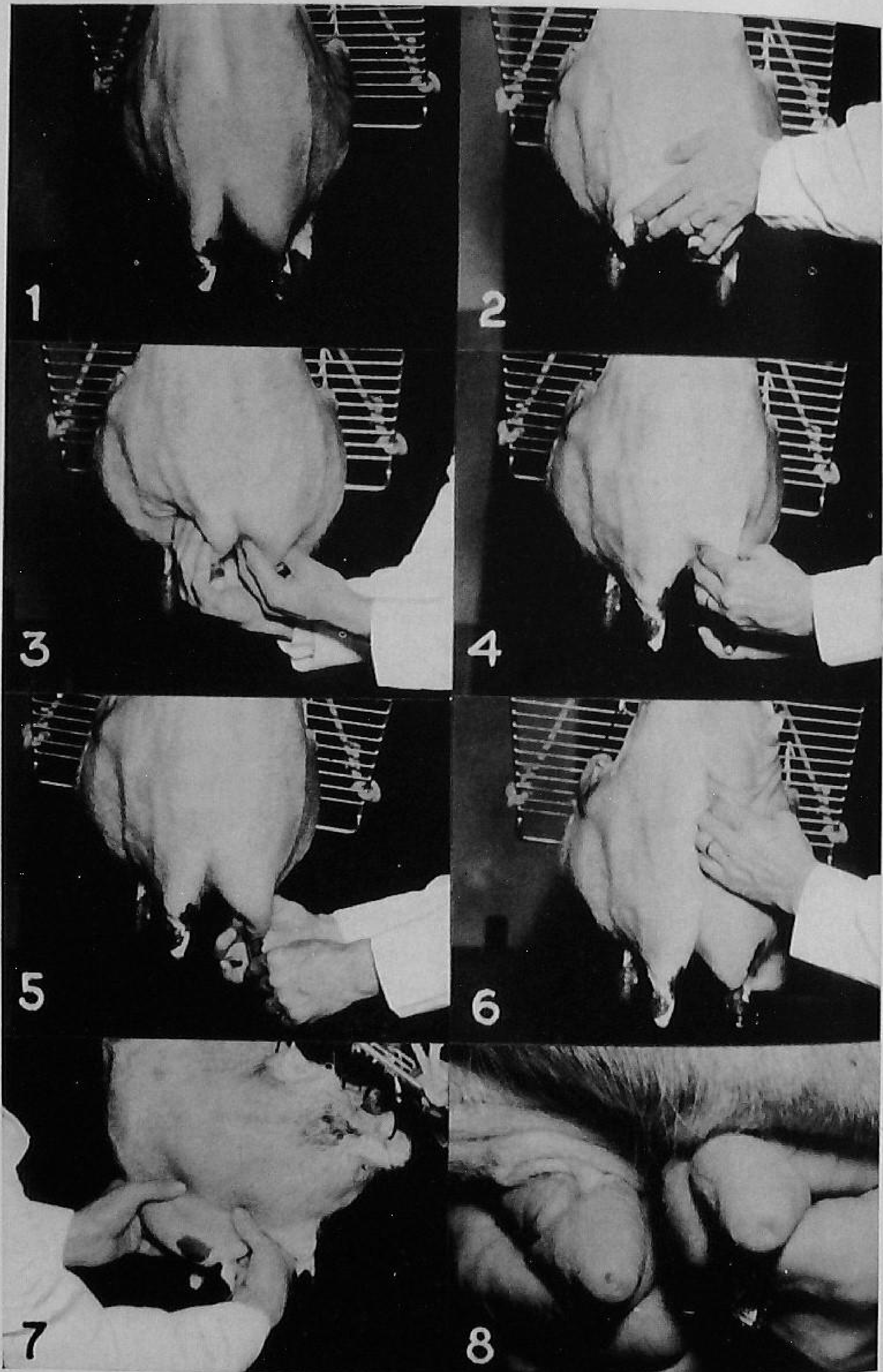

Figure 107

diseases, the status of the organ can be determined by the nature and extent of the *pathological changes in the tissues.*

The symptoms of mastitis vary widely in detail, but if one follows individual cases through successive lactations the chief manifestations prove to be uniform. As a rule the onset is chronic, though it may be acute, and as the tissue changes develop there are active and latent periods varying somewhat according to the methods of feeding and milking. The condition may be unsuspected until a routine examination of the milk and udder reveals abnormal milk and fibrosis. There may be a history of a previous acute attack, perhaps at the time of freshening, or in some previous lactation period. Often there is a report that the milk production is low, or that the cow is a "short milker." Diseased cows may have been purchased. If the milker has been observant, there is a record of "garget" or flakes in the milk.

When the initial attack is *acute,* the normal flow of milk may suddenly discontinue and be replaced by a little serous fluid. Heat, pain, and swelling may or may not develop. Occasionally acute general symptoms are present; they may be marked, and in rare instances death occurs. In a less acute form, milk from one or more quarters contains flakes and clots, often appearing first from one teat and then from another until all four are implicated. Under proper treatment the acute and more obvious changes in the udder and milk usually recede within a week to ten days and the udder becomes "apparently normal," the case is "cured." Unfortunately a complete cure is not the rule, yet such a result is possible, in a clinical sense, even though a circumscribed induration may permanently remain. Recognition of these acute or subacute conditions is seldom difficult, though acute mastitis has been diagnosed as indigestion because of atony of the rumen and failure to eat.

In a group of mastitis-free cows there may be occasional acute attacks, while in other similar groups such incidents may be rare. In cases where the udder has been entirely normal until the time of the initial acute attack, the onset is sudden, the udder is swollen and painful, and the milk is abnormal; but inoculation of blood agar plates with milk may give entirely negative results. Recovery of such cases is usually complete under proper treatment. Acute mastitis is infrequent in herds that are relatively free from the chronic form, and such attacks run a more favorable course than in highly infected herds.

Far more significant than these acute attacks that appear irregularly, either as a first or a second or any other flareup, are the number of udders that show distinct or marked indurations (fibrosis) as revealed by physical examination of the milked-out udder—*chronic mastitis.* This

condition has been variously named, as cured, latent, occult, and subclinical, terms which usually mean that the disease is not obvious to the casual observation of a layman, though often recognized by the owner or milker. Control of this condition is of increasing importance because of frequent exclusion of milk from such udders from market, and because owners often appeal for relief from a disease that is gradually destroying their income. The degree of mastitis is in almost direct proportion to the degree of induration or fibrosis. Slight changes, however, are apparently of no significance. An initial mild attack may leave the udder normal, or it may leave a circumscribed induration that apparently remains unchanged and nonprogressive. Slight indurations not originating from an acute inflammation may fail to progress. After the udder has acquired a group 3 or 4 classification, however, the lesions tend to become more extensive from one lactation period to another. Where intensive feeding and unsanitary milking are practiced, the disease may progress rapidly. When advanced cases of mastitis are segregated, the milder forms appear to become less active. Evidently this separation prevents repeated new infections.

On the basis of complete clinical records of many cases in different herds we have proposed a classification of quarters of udders as normal, slight, distinct, and marked, according to the degree of fibrosis; and of udders as 1, 2, 3, and 4, in respect to all of the clinical evidence obtainable. In general, a No. 1 or 2 udder is regarded as normal, while Nos. 3 and 4 are affected with mastitis and subject to segregation where control measures are adopted.

Physical Examination of the Udder.—The work is best conducted directly after milking. An examination is not satisfactory when the udder is distended with milk or when it is swollen and congested before and after freshening; the elasticity of the glandular tissue cannot be recognized.

An outline of methods of observation and palpation of the udder is illustrated in Figure 107.

(*1*) Observe the rear quarters for the presence of atrophy, and for variations in the size and position of the teats.

(*2*) Observe the front teats for atrophy by raising rear teats backward and upward; note whether the ends of the teats are in the same horizontal plane.

(*3*) Palpation of the cysterns. Raise the quarters together simultaneously to compare the consistency and weight by lightly raising and palpating the tissues in the region of the cysterns with the finger tips. There may be a distinct variation in the weight, firmness and surface which

reveals fibrosis in one as compared with a normal soft glandular structure in the other. Variations here suggest the probable findings throughout the quarter.

(*4*) and (*5*). Method of locating circumscribed areas of *fibrosis in the ventral part of the quarters*. Grasp, but do not stretch, the teat with one hand and compress the tissues with the other. In a normal, elastic quarter the opposing thumb and fingers are separated chiefly by the opposing layers of skin. In fibrosis they are separated more or less by a firm mass. The degree of fibrosis is estimated by the difference between the two hind quarters and the two front quarters both anterior and posterior to the teats. But do not compare front quarters with hind quarters.

(*6*) *Superficial palpation* of the surface. With light compression the skin is raised in folds while the finger tips glide over the underlying surface to detect the degree of smoothness.

Each quarter may be normally smooth or normally lobulated. A normally lobulated surface is uniform in contour and if projections are present they are small and regular, while a pathological lobulation is not uniformly distributed and the lobulations are not uniform in size. When the thumb and finger pass over the surface of one quarter the subcutis may present a smooth surface while the corresponding surface of the mate to this quarter may be lobulated, indicating fibrosis.

(*7*) *Palpate for asymmetry*. Grasp each quarter firmly with both hands, which are moved back and forth firmly under firm compression, noting lobulations and consistency. The tissues are alternately compressed and relaxed. The degree of atrophy and consistency is also judged by lifting each quarter with both hands and comparing each quarter with its mate on the opposite side for weight, elasticity, and size. Coarse irregular lobulations are of greater significance than fine lobulations uniformly distributed. When a difference in size is found, as one large and soft and one small firm front quarter, the smaller one may have been affected with an acute mastitis that caused suspension of the milk-flow and shrinking of the glandular tissue; an exact estimate of the degree of injury may not be possible until the next lactation period. If the smaller quarter is found to be distinctly nodular, however, instead of smooth and regular on the surface, one may conclude that connective tissue has replaced glandular tissue; it has become indurated. During the active stage an infected quarter may be considerably larger than its mate—one hind quarter is decidedly larger than the other, and doubt exists as to whether one is swollen and indurated to a pathological degree, or whether the other is atrophied to a pathological degree. As

a rule the question is answered by the presence of irregular indurations in the affected quarter, or by the reaction to a bromthymol-blue or chlorine test of the milk.

(*8*) Examination of the ends of the teats for patent meatus (Fig. 107 (8), teat at the right), relaxed sphincter, and ease of milking.

The significance of induration depends on the degree and distribution of fibroses in different quarters. Circumscribed indurations may be single or multiple and large or small, while diffuse indurations may involve an entire quarter. Their presence may be so slight as to have no significance, or so extensive as to ruin the cow for milk production. A quarter is termed *slight* or *suspicious* when it contains not more than one or two circumscribed lesions not over 2 inches in diameter, or when there is a slight lack of uniformity in the size and consistency. A quarter is termed *distinct* when it contains an induration approximately 3 inches in diameter, or when there is a distinct difference on superficial palpation only, or when distinct atrophy with indurations is present. A quarter is termed *marked* when the indurations are diffuse and extensive, or circumscribed and multiple. A quarter is not designated as marked if it consists largely of elastic tissue. Numerous circumscribed indurations in large udders with an abundance of elastic tissue would give a distinct rather than a marked classification; this condition is frequent in old cows. Diffuse indurations in all four quarters may give to a badly diseased udder an "apparently normal" appearance.

There is no distinct line of demarcation between these three groups, and the legend S+ or D+ is used for border-line cases. Those who acquire skill in examination of the udder reach uniform decisions in respect to classification.

The findings on each quarter are recorded as follows:—

—, the quarter is entirely normal.

S, fibrosis is slight or suspected. There are single indurations not more than one or two inches in diameter.

D, fibrosis is distinct; Da, with slight atrophy, DA, with marked atrophy; Da lob, with coarse lobulations; D+A lob P, very distinct with marked atrophy, coarse lobulations, and relaxed sphincter. There is multiple fibrosis or a single induration approximately two to three inches in diameter. P, patent teat meatus.

M, fibrosis is marked. Disseminated or diffuse extensive indurations have largely replaced the glandular tissue.

The plus sign indicates an intermediate degree, as S+, D+. Udders of first-calf heifers are normally firm, and the individual quarters are sometimes marked D. With sufficient practice, one may be able to classify udders of dry cows. When the udder is congested, tense, and

edematous in relation to parturition, it is not suitable for examination. If an udder is edematous, or "doughy," unrelated to parturition, it is severely judged.

Classification of udders is made on evidence obtained in the stable by physical examination of the udder. In badly diseased herds interpretations should be made more severely than in well-managed herds with light infection.

Number 1 is normal in every respect or contains a single slight induration in each quarter or single slight indurations in either the front or hind quarters, being uniformly symmetrical. There are comparatively few Number 1 udders. The milk is normal.

Number 2 contains a few slight indurations (S) or a single distinct lesion (D). If two quarters are marked distinct and the milk gives no reaction to the bromthymol-blue test, the classification would usually be No. 2. The milk is normal (not green) to a bromthymol-blue test made within 24 to 48 hours after calving. Occasionally, there may be a transient increase in alkalinity.

The following table contains legends applied to No. 2 udders as determined by physical examination:

		Quarters				
		LH	LF	RH	RF	
1	Phy	Da	S	S+	S	
2	Phy	D	—	D	—	
3	Phy	Da	D	S	S	
4	Phy	S	D	S	S	
5	Phy	D*	S	S+	S+ 2+	*Staphylococcus*
6	Phy	D+	S	S+*	D*	*Staphylococcus*
7	Phy	*D*	*D*	*D*	*D*, first-calf heifer.	
8	Phy	S+	S	Da lob	S 2+	
9	Phy	S+	S	D	S+	
10	Phy	S	S	S+a	S+A	

Number 3.—An udder is marked No. 3 as follows:

(a) When there is distinct fibrosis (D) in two or more quarters combined with either atrophy, or a bromthymol-blue reaction of the milk; often both are present.

(b) When there is extensive fibrosis (D+) in a single quarter. Often this is combined with lobulations (lob), atrophy (a or A), a change in the color and consistency of the milk (dc) as seen in a test tube, and an abnormal teat sphincter—fibrous thickening or scar or patent teat orifice (p) or stenosis (spraying when milked).

The following table contains legends applied to No. 3 udders as determined by physical examination:

		Quarters				
		LH	LF	RH	RF	
1	Phy	Da	D+	D	D+*	*Str. agalactiae
	BB	—	—	—	—	
2	Phy	DA*	—	D	S+	*Str. agalactiae
	BB	slg	—	slg	slg	
3	Phy	Da	D*	D	Da*	*Str. agalactiae
	BB	lg	slg	lg	lg	
4	Phy	Da*	S	D*	S	*Str. agalactiae
	BB	G	—	lg	—	
5	Phy	Da	S	D+*	D	*Staphylococcus
	BB	slg	—	lg	—	
6	Phy	D	—	D+**	D*	*Str. agalactiae **Staphylococcus
	BB	slg	—	G	lg	
7	Phy	S+	—	S+	D+P*	*Staphylococcus
	BB	—	—	—	G	
8	Phy	S+	D+a*	S+	S+	*Staphylococcus
	BB	—	dkG		—	
9	Phy	D+	D	D+	D	No infection
	BB	—	—	G		
10	Phy	D+*	S	S+	S	*Str. agalactiae
	BB	—	—	—		

Phy, physical. BB, bromthymol-blue reaction

There may be a history of repeated attacks. Production is usually good. Occasionally the milk from the affected quarter shows flakes or clots, is serous or watery, and reacts to the bromthymol-blue test. There may be a distinct change in the character of the milk in a period of two to four weeks. If the udder is distinct in three quarters, the elastic tissue abundant, and the milk usually normal it may be placed in this group. If the udder is distinct in one quarter, slight in the other three, and the milk is usually abnormal (case No. 7), it would be classed at 3+ or 4.

In routine physical examinations, one may classify udders without an examination of the milk. The appearance of the milk, and the bromthymol-blue reaction fluctuate from time to time, while the degree of fibrosis is either constant or progressive. Apparent exceptions occur

when a subacute inflammation recedes and the affected quarter becomes more pliable.

Number 4.—*Fibrosis* is distinct and multiple or marked and diffuse. Atrophy and asymmetry are common, but when all four quarters are affected the udder may be well-balanced. If the lesions are marked (M) in two quarters, the udder is classed as 4, even if the milk is normal. If a quarter leaks freely the udder is classed as 4. There is a history of repeated attacks and the production is below normal; usually it is low. The *milk* usually contains flakes, clots, or is watery. Bromthymol-

Quarters

		LH	LF	RH	RF	
1	Phy	Da	MA	D	D	
	BB	—	dkG*	—	—	*pus
2	Phy	D+*	D+**	D+	D+	**Str. agalactiae*
	BB	—	—	—	—	***Str. dysgalactiae*
3	Phy	D+p*	Dp*	Dp*	D*	**Str. agalactia*
	BB	slg	—	lg	—	
4	Phy	M	D*	D**	Da**	**Str. dysgalactiae*
	BB	—	lg	lg	G	***Str. agalactiae*
5	Phy	D	S*	M**	S*	**Str. agalactiae*
	BB	—	lg	dkG	lg	***Str. dysgalactiae*

blue gives a positive reaction. Occasionally in a high producing cow the milk may be apparently normal during the first two or three weeks of lactation, but usually there is a distinct reaction during this period.

A classification based upon a single survey will contain a few individuals in the 2+ or 3+ group. But after a few examinations of the herd one is usually able, through comparison of the indurations with milk reactions and production, to classify the cow definitely. This method should be interpreted as a general guide, and not as a list of specifications in which a formula for each udder may be found. Arranged in order of frequency of occurrence, the evidence may be listed as follows: 1, physical examination of the udder, including the history and milk production; 2, chemical and physical examination of the milk; 3, number of leucocytes; and 4, bacteriological findings.

This classification makes it possible to protect normal cows against exposure to infection. Comparative examinations show that No. 4 udders are eliminating infection almost constantly, No. 3 udders are eliminating it less frequently, and that milk from No. 2 udders rarely shows

bacteriological evidence of mastitis. While milk from Nos. 3 and 4 may be free from infection at times, this finding does not change their status.

Examination of the Milk.—Much information may be obtained by an examination of the milk in the stable, and this may be supplemented by the cultural and smear examinations previously described. *Strip cups* are in common use in herds producing certified milk and they have been adopted to some extent by others where mastitis control measures are in operation. A pint tin cup or larger receptacle is fitted with a removable top about 1¼ inches deep. One half of the bottom of this section is made of fine wire mesh, 100 squares to the inch, the other half is made of tin. Fine black cloth may be used for the same purpose, and there are various forms of strip cups for sale by dairy supply firms. The strip-cup test is made when the udder is filled with milk, just before the regular milking time. Draw the first two to four streams from each teat directly on the sieve and examine for flakes or clots. The top should be removed and rinsed in water after each test in order to remove the thin layer of foam that may obscure a slight flakiness. Routine use of the strip cup is of great value in revealing the first stages of an acute inflammation, whether it be an initial attack or a flareup of a permanently diseased udder; the case is discovered early when segregation and treatment may be most effectively applied. A single survey of a herd with a strip cup for the purpose of detecting cows affected with mastitis is not of great value, since a large per cent of the cows distinctly affected with chronic mastitis give milk that is usually free from flakes or clots. The presence of flakes is commonly regarded as an indication of mastitis, but they may be present in milk of an abnormal color as a transient condition after an injury. Milk may also present a change in color and consistency, becoming watery or lighter in color, or yellowish, and such milk may be free from flakes as shown on the strip cup. It may be recognized in test tubes by comparing it with normal milk, or by allowing a small stream to flow over a glazed black surface. For this purpose a sheet of bakelite tilted lengthwise in a small rectangular baking tin painted black is excellent, and it has largely replaced the tin cup with a wire mesh bottom. Milk drawn upon the glazed black surface may show a slightly changed color (dc) or a distinct change from the normal (DC) or it may contain flakes and clots.

Examination of the Udder Secretions of Dry Cows.—Physical examination of the udders of dry cows is often useful in the detection of mastitis; especially at the beginning of the dry period or before enlargement due to approaching lactation. Swollen firm quarters may contain pus and yield *Str. agalactiae.* Prompt treatment and resumed

frequent milking of such quarters may prevent an active severe attack when the cow freshens. Routine examination of all dry cows, including the udder secretions, is of great value in the herd control of mastitis. An examination of the secretions of the udder when the cow is dry may be more instructive than an examination of the milk during lactation. This observation is of special value in the inspection of dry cows for purchase.

At the beginning of the dry period the secretion should be uniform in color and consistency, and free from clots or flakes, or any resemblance to pus. An infected quarter may contain pus after milking has been discontinued. This may increase in amount during the dry period, be quite abundant when the cow freshens, and result in a badly infected udder. About six weeks after milking has been discontinued, the milk of a normal udder has the appearance of honey; it is clear, amber in color, and free from turbidity or sediment. Secretion from a quarter affected with staphylococcic infection is often turbid and milky. In streptococcic infection the secretion may be milky and contain sediment. Laboratory examination of the secretion from a dry quarter reveals the absence or presence of infection, as it does in the milk during lactation. The amount of the secretion is variable.

Keeping Qualities of the Milk.—The keeping qualities of the milk held at a temperature of 37°C. for 24 hours gives useful information on the health of the udder. Samples are collected from each quarter in sterile glass cylinders 4 inches high by 1⅛-inch diameter, provided with sterile rubber stoppers (any small sterile glass container is suitable). The sample is taken as for bacteriological examination, after wiping the teat with cotton moistened with alcohol. Normal milk kept in this manner shows a definite milk and cream line; when the tube is inverted the bottom and sides are free from sediment. In mastitis milk, there forms a layer of yellowish serum (whey) between the cream line and the milk, the milk itself may appear watery, and sediment is deposited on the glass. Under the influence of heat these changes may occur as early as six hours, while normal milk may appear normal after forty-eight hours. When incubated milk from a normal quarter is returned to the refrigerator it may retain its normal appearance for as long as two weeks. In the hands of the inexperienced this test is of more value than the bromthymol-blue test, since it is not apt to be misinterpreted.

The *alkalinity* or *pH*, is revealed by the bromthymol-blue (thymol-bromol) test. Mastitis milk is usually alkaline in reaction and salty in taste. In Switzerland it has long been a routine practice to apply the taste method when an udder is being examined. According to Minett the reaction of milk is normal after about the ninth day. Our observa-

tions shows that when the udder is normal, cows 1 and 2, the bromthymol-blue test usually gives a normal yellowish green reaction as early as 24 to 48 hours after calving, and that the alkalinity is regularly normal on the first day. In chronic mastitis, milk from the affected quarters often gives a green or light-green reaction immediately after calving. Apparently an increased alkalinity at this time suggests mastitis, and the time required to return to normal may suggest the degree of inflammation. In Nos. 3 and 4 this reaction may persist for one or two months and even longer. The milk from a normal udder may infrequently show a transient high pH in the middle of the lactation period, and in about the same frequency flocculent milk from a diseased quarter may show a normal pH. In general, however, a distinct green reaction indicates mastitis. The bromthymol-blue test gives information when its results are compared with other manifestations. The various degrees of reaction are slight light green, light green, green, dark green, and orange (acid reaction). With few exceptions the color reaction in mastitis is variable in the milk from the different quarters. Some have been confused because of a reaction in milk that failed to show infection on bacteriological examination, while others have been equally confused because of reactions where there was no "apparent mastitis." A complete clinical examination supports the evidence of a persistent bromthymol-blue reaction. There is a tendency to forget that in every case of mastitis the milk varies from time to time, and that its chemical and bacteriological manifestations are equally variable. There is no reason why chemical and bacteriological tests should both be positive or negative at the same time. The seat of the disease is in the udder, rather than in the milk, and the significance of the reaction of any test of the milk relates to lesions in the udder rather than to some other test.

Bromthymol-blue solution is prepared by dissolving 1 gram of bromthymol-blue powder in 500 cc. of 47.5 per cent alcohol. To adjust it to an alkaline range, add about 1.5 cc. of a 5 per cent sodium hydroxide solution. If this proves to be too much, the addition of a few drops of a 5 per cent solution of hydrochloric or sulphuric acid brings it back to the neutral range. It is important that this indicator be only slightly alkaline or slightly acid. An acid bromthymol-blue solution is red, while an alkaline solution is green. If the solution is too acid it may not give a distinct green in alkaline milk; if it is too alkaline it may impart a green color to normal milk. An equally satisfactory solution is made by dissolving 1 gram of bromthymol-blue in 160 cc. of one-hundredth normal sodium hydroxide solution and adding enough distilled water to bring the whole volume up to 750 cc. Milk for examination is drawn after discarding the first four or five streams of the

foremilk—that which is drawn into the strip cup. Then from each teat to be tested draw 5 cc. of milk into a test tube and examine the contents for any difference in color or consistency. A slightly watery or discolored milk is easily recognized if the comparison is made in good light. The test is made by adding 0.5 to 1 cc. of the indicator to 5 cc. of milk. Normal milk gives a slightly yellowish-green or greenish-yellow color. The degree of reaction depends on the degree of alkalinity above the normal of 6.2 to 6.5.

The *chloride test* is regarded by many workers on mastitis as the most accurate indicator of the presence of fibrosis. Normal milk contains 0.09 to 0.14 per cent chlorides. A field chloride test for the detection of mastitis has been developed by Hayden. This reveals the presence of chloride when the percentage is 0.14 or over. Hayden's[20] test is as follows: *Reagents:* Dissolve 1.3415 grams of C.P. silver nitrate in 1 liter distilled water. Make a 10 per cent solution of potassium chromate in distilled water. *Procedure:* Measure an accurate 5 cc. of the silver nitrate solution into a test tube. Add 2 drops of the chromate solution. A red color develops at once. Add an accurate 1 cc. of milk to this combination. A yellow color will develop in one minute or less if the chloride is 0.14 per cent or over. The yellow color develops very rapidly if the chloride is high. The red color will be maintained if the chloride is under 0.14 per cent.

The *Hotis Test.*—This is a highly accurate selective test for the detection of *Str. agalactiae* in milk from infected quarters. It was described in 1936 by Hotis and Miller.[21] The test consists in adding 0.5 cc. of 0.5 per cent bromcresol purple to 9.5 cc. of aseptically drawn milk. Milk containing mastitis streptococci shows a typical change after 24 to 48 hours incubation at 37°C. Murphy[22] has reported that the "application of the test to 753 samples of milk in conjunction with cultural examination in blood agar showed them to be in perfect agreement for 95 per cent of the samples. . . . It was found that two classes of reaction are characteristic for the presence of group I mastitis streptococci (*Str. agalactiae*)." This characteristic reaction consists of a thick yellow deposit in the bottom of the tube, or yellow colonies adhering to the sides of the tube. Since it requires the use of an incubator it is a laboratory rather than a field test.

Diagnosis.—In the diagnosis of mastitis, the history, the condition of the udder and milk, and any other symptom that may be obtainable, determine the classification. Our experience indicates that it is possible to make an accurate diagnosis by means of either a clinical examination made in the stable, or an examination of the milk in the laboratory. This is contrary to a commonly expressed opinion, repeated by Seele-

mann,[5] who writes that frequently a quarter may contain streptococci and show abnormal secretion when changes in the tissue cannot be felt, even by means of a thorough examination. This point is of great importance in relation to mastitis control. The intermittent elimination from the udder of bacteria causing mastitis requires that examinations for their detection be made frequently. And segregation of cows by this method alone is expensive.

The question may well be raised, whether the modern laboratory workers who have decided that clinical methods applied by Bang, Nocard and others are not effective are qualified to make such examinations and decisions. While the technic may appear to be simple, skill in the classification of udders is acquired only after long practice. It includes not merely the art of detecting indurations, but experience and judgment in reaching a decision upon all the evidence obtainable.

In making a decision, consider that the indurations are permanent or progressive, and that abnormalities in the milk vary in degree and persistence, particularly in groups 2 and 3. In these groups chemical (*p*H, chlorine) and cellular (leucocyte) changes are often present when bacteriological findings are negative. After the lesions are advanced the milk, with few exceptions, is permanently abnormal. First learn the age, last calving date, and any previous history of mastitis or "garget." Mastitis is one of the most frequent causes of low milk production, and when an owner reports that a cow is a short milker or a low producer, it should be suspected. Yet a high-producing cow with a large udder containing several distinct lesions may still milk heavily if the indurations are circumscribed.

Methods of Control.—In the control of mastitis one is concerned with the two major causes mentioned under etiology: the infected cow and protection of the udder. According to one view mastitis control depends on identification of the infected cow by means of bacteriological examination of the milk, just as tuberculosis control depends on identification of the infected cow by means of the tuberculin test. This method of diagnosis is desirable but it is not generally available, and it is not essential.

It has been demonstrated in many herds that segregation of the clinical forms of mastitis in combination with hygienic milking and stable hygiene will promptly check the disease, as reported many years ago by Bang. There is considerable evidence in support of the view that protection of the udder is of even greater importance than the diagnosis. Many new cases begin with injury to a teat from which infection eventually develops in all four quarters. In our small experimental herd where every second cow was heavily infected with *Str. agalactiae*,

and no special precautions were taken, only one new case developed in three years, without previous injury. One can at least claim that the few infected udders which do not fall in class 3 and 4 will not be a source of contagion where cleanliness and disinfection are practiced. Under these conditions infection does not readily spread between infected and noninfected individuals in the class 3 group. The badly diseased class 4 cow is the chief source of infection and this animal is always a low producer.

The minimum requirements are adequate space, stall partitions to prevent treading on the teats, and a well-bedded dry floor. The use of superphosphate on the floors and gutters, but not on the stall-beds, is desirable; it absorbs moisture and is a mild antiseptic.

All affected cows should be placed together and permanently *assigned stanchions* according to the degree of mastitis. Thus, all in No. 4 group would occupy a definite part of the stable and never enter other stanchions. In the same manner the No. 3 cows would be together. Nos. 1 and 2 are regarded as a single unit, and not stanchioned among either Nos. 3 or 4. It is probable that infection spreads chiefly from No. 4 cows, but all animals classed as 3 or 4 should be tied separately from the 1 and 2 class, regardless of the history or the appearance of the milk. This may be difficult to accomplish when an owner is unconvinced that a favorite cow is abnormal, or when he decides that a No. 4 cow is "cured." Under usual conditions the spread of infection from cow to cow is gradual, but finally 50 per cent or more of the udders may show distinct or marked fibrosis and the herd is badly damaged. The most desirable segregation is provided by separate stables and milkers, but this is seldom practical. Sufficient isolation is provided when all groups occupy the same stable in separate units, if the normal animals are milked first.

The routine use of a strip cup informs the milker of acute cases and of recurrent activity in time to apply effective treatment and prevent the spread of infection; it also assists in the classification of udders. When a cow shows watery milk, flakes, clots or pus repeatedly, she should be removed from the milking line, particularly if she stands in a clean group. In the treatment of such cases the grain ration is reduced, and the quarter infused with an antibotic (2 million units penicillin; 20 cc. Penicle, and ⅕ to 1 Gm. streptomycin).

In washing the udder, one uses a special single-service 10¾" x 11" paper towel ("Kotowl"). The operator wrings the towel partly dry from a chlorine solution of 200 parts per million, or from soapy water at 130° F. prepared from soap flakes or Ivory soap, washes the udder, and discards the towel. A few streams of milk are withdrawn from each

teat, and the machine is applied directly thereafter. The operator prepares the udder for milking about two cows ahead of the machine. While washing of the udder is desirable, if it can be done properly, the "let-down" of milk may be accomplished by stripping ahead of the milker without washing. It is commonly advised that final stripping of the udder be done with the machine, and that a milking machine should operate in a minimum of time in order to avoid injury to the teats. If the cow is hand-stripped it is obvious that the time occupied by the machine can be reduced, and consequently it is recommended that hand-stripping be practiced. While complete milking of certain individuals may be accomplished in the minimum time requirement of the machine, there are numerous exceptions. A variable amount of milk may remain in one or more quarters, and a number of experiments have shown that any degree of mastitis is aggravated by incomplete milking. Furthermore, strippings are high in fat, and their loss results in a diminished butter-fat test for the herd.

The milking machine should not be used on obviously diseased quarters; they should be milked by hand. Under average conditions, mastitis infection spreads less rapidly in herds milked by hand than where a machine is used. But a milking machine is safe if the equipment receives proper care and is not used upon diseased udders. If it is impractical to restrict the use to normal cows, the milking cups should be cleansed after each cow is milked. A method that seems effective is first to rinse them in a pail of clear water and then in a chlorine solution of 200 p.p.m. Objection has been raised against the common practice of dipping the milking cups and teats, because such exposure to a chlorine solution of 200 p.p.m. leaves infection which has been revealed by laboratory examination. Failure to attain absolute sterilization of the skin or of all parts of equipment has not led surgeons to abandon the elementary principles of cleanliness and disinfection, and whatever theory one may hold concerning the cause of mastitis, it is a common observation that losses are curtailed where cleanliness and disinfection are practiced. For information on sterilization of milking machines, see Bul. 492, State College of Agriculture, Ithaca, N.Y.

In hand milking it is desirable that the milker thoroughly wash his hands after each cow is milked. Before milking each cow a milker should wash his hands with soap and water, disinfect them with a chlorine solution (100-200 p.p.m.) and wipe them dry, preferably with paper towels. Do not milk on the floor, and do not permit wet milking.

For dipping teats only a small amount of chlorine (½ to 1 pint, having a strength of 200 p.p.m.) is necessary to dip the ends of the teats after milking. Dip the teats of 20 to 30 cows and discard the solution.

Provide a basin constructed preferably of graniteware, porcelain, enamelware, or glass and large enough to accommodate the four teats of an udder at one time.

Once a week the litter should be removed, the surface of the platform scraped, and sprayed with a disinfectant (hot lye 3 to 5 per cent solution, chlorine solution, 400-500 p.p.m.). Provide an adequate stall-bed, partitions for each cow, and plenty of bedding. Heavily producing cows should be milked by hand and preferably three times daily. First-calf heifers and fresh cows from the clean group may be stanchioned with the clean group if the milk is normal on the strip cup and the bromthymol-blue test. Additions from other herds should be quarantined until found to be normal.

Treatment.—The treatment of chronic mastitis presents two problems: (1) The group of cows with udders that are damaged, but still suitable for milk production; and (2) the cow with active mastitis, and yielding flakes or clots.

The damaged group contains only Nos. 3 and 4. These should be so arranged in the stanchions that the milker passes from the better udders to the more advanced cases. Special efforts should be made to practice complete milking of affected quarters. More than the usual care is necessary when the cow is being dried off, and certain individuals cannot be dried off without causing an acute flareup; such cows should be milked continuously. The grain ration should not be high. On a restricted diet and careful milking, such a group may produce fairly well, and the less advanced cases may improve. Forced feeding to produce maximum production, however, may result in disastrous acute activity.

Vaccination has been widely practiced, but there is no evidence that it exerts either a curative or a prophylactic effect.

Acute mastitis responds to treatment when the udder has previously been normal, but acute septic mastitis with a high temperature and severe general symptoms may be fatal. In a series of 54 quarters affected with acute mastitis, 22 were infected with coliform organisms, 8 with hemolytic staphylococci, and 9 with green streptococci (*Str. dysgalactiae* and *Str. uberis*)—Ferguson.[23] In a subsequent series, hemolytic staphylocci were predominant.

Since the administration of antibiotics, the mortality from acute septic mastitis has been materially reduced. While the details of medication vary somewhat, the following procedure is an example. Previous to infusion of the udder administer 5 cc. of posterior pituitary extract intravenously to stimulate immediate flow of milk, and then milk as long as any can be obtained in the next fifteen minutes. Then infuse the quarter with 500,000 units crystalline penicillin and ½ to 1 Gm. strep-

tomycin disolved in 50 cc. distilled water, or saline solution, or an aqueous solution of either sulfamerazine or sulfamethazine. Apply an ice pack supported by a suspensory bandage. Administer intramuscularly 1 to 3 million units of penicillin in oil, plus ½ Gm. streptomycin, and give two 1-ounce capsules sulfamerazine orally, followed by one every twenty-four hours. The penicillin may be repeated daily.

Disinfection of the Mammary Gland.—For a number of years the treatment of *chronic mastitis* by injection of disinfectants through the teat canal into the glandular tissue, chemotherapy, has been practiced. In 1934 Steck[24] reported that "chemotherapy is a useful weapon in the control of agalactiosis, and even the cases that cannot be cured completely may be rendered less dangerous through the considerable reduction in the number of germs spread."

In practice, treatment of mastitis is usually applied to cows in lactation following the appearance of flakes, clots, or pus in the milk, and without identification of the type of infection. While such identification is desirable, experience shows that prompt treatment as soon as the symptoms appear is most apt to be effective,[25] and that frequently when post-treatment samples of milk from such cases are examined in the laboratory no infection is found; this is especially true of heifers and young cows with no history of mastitis. Furthermore, a number of veterinarians have reported success in the treatment of mastitis diagnosed by physical examination only, and without laboratory examination of the milk. We have observed instances of improvement of the milk following treatment of dry quarters from which the secretion during lactation yielded no significant bacteria or no bacteria whatever. Treatment of udders during the dry period is especially effective; this may include quarters from which milk samples yield pathogenic organisms, or udders with a history of recurrent mastitis during the previous lactation, or upon examination when dry yield abnormal colostrum and have fibrosis. Whenever available, treatments of dry udders should be guided by the results of laboratory examinations.

In the selection of a disinfectant it is desirable to use one that does not irritate the udder, nor cause prolonged change in the quality of the milk. These conditions are found in several of the recently introduced antibiotics. One of the most widely used and effective combinations is penicillin and streptomycin in Penicle, a water-in-oil emulsion, or similar vehicle. Examples are, procaine penicillin G in aqueous suspension, 100,000 units; Penicle, 20 cc.; streptomycin 1/5 Gm. (100-20-1/5). Or in more severe attacks: procaine penicillin G in aqueous suspension, 500,000 units; Penicle, 40 cc.; streptomycin, 1 Gm. (500-40-1). In active lactation this may be repeated every twenty-four hours; in dry

infected udders it may be given once or more according to the degree of mastitis, or the type of infection (staphylococcus, pseudomonas).

The first obvious result of treatment is improvement in the quality of the milk, or absence of infection from post-treatment samples. Infection may disappear within a few days or weeks or the bacteria may gradually recede over a period of months, as in some cases of staphylococcal mastitis. *Str. agalactiae* infection may be overcome by treatment only to be followed by staphylococcus, and staphylococcus is often replaced by *Str. agalactiae*. After infection in a quarter has been overcome there remains a tendency to reinfection, and even when this does not occur the milk may remain abnormal. This tendency to recurrent infection, and to a failure of the milk to return to normal, is often observed in quarters with a D+ fibrosis or a marked atrophy. Improvement in the quality of the milk, as well as freedom from infection, is favored by a long dry period. In severe types of mastitis the treated quarter may atrophy and almost cease to function.

It is desirable that one identify the type of infection in each case, but facilities for this important information are not available to all veterinarians. If our observations and opinions are correct, an accurate knowledge of the physical condition of the diseased quarter is more important, in estimating the prognosis, than a knowledge of the type of infection. And this information is available to all. In the classification of udders, there is no great difficulty in learning how to identify slight, distinct, or marked fibrosis in each quarter and in noting the degree of atrophy.

In the treatment of mammary infections with the more refractive staphyloccus, Schalm and Wood[31] cured 90 per cent of lactating and 73 per cent of dry quarters with infusion of 1 million units penicillin and 1 Gm. dihydrostreptomycin in 50 cc. water administered four times at 24-hour intervals. Terramycin, 7.5 Gm. ointment in a collapsible tube, infused into the nonlactating udder, in a limited experience, suggests efficiency in clearing both staphylococci and streptococci.

RECOMMENDED TREATMENTS FOR BOVINE MASTITIS

New York State Mastitis Research and Control Program
New York State Veterinary College
Cornell University, Ithaca, N.Y.

	LACTATING COWS	DRY COWS
Clinical Mastitis—Chronic or Mild		
Known *Streptococcus* Known Hemolytic *Staphylococcus* Unknown Infections.		
Penicillin[1]—200 or more T.U. + ⅕-½ gm. dihydrostreptomycin in water-in-oil emulsion.[2]	Repeated as indicated depending on severity of case.	Same.
Procaine Penicillin—200 or more T.U.[3] + ⅕-½ gm. dihydrostreptomycin in peanut oil commercially prepared. (Some may contain sulfas.)	10-20 cc. (or more) 1 to 2X at 48-72 hours.	Same.
Penicillin[1]—200 T.U. in 50-80 cc. aqueous sulfonamides.[3, 5]	2 to 4X at 12-24 hours.	Not recommended.
Terramycin—400 or more mg. in ointment tube.	1 to 2X at 24-48 hours, or repeated as indicated.	Same.
Neomycin—0.5 + 100-200 T.U. penicillin in water-in-oil emulsion.	Repeated as indicated.	Same.
Tyrothrycin—20-40 cc. in oil or in other mixtures.[4] Can combine with 200 or more T.U. penicillin.	Not used unless "drying off" quarters.	Must have some secretion in udder. Milk out after 48 hours if swelling persists.
Known Streptococcus agalactiae, with or without clinical symptoms		
Penicillin[1]—100-200 or more T.U. in 10-20 cc. water-in-oil emulsion.[2, 3]	1 to 3X at 24-72 hour intervals depending on severity of case.	Generally 1X 7-10 days after drying off. May repeat when springing.
Aureomycin ointment—400 or more mg. tube.	1 to 3X at 24-48 hours.	1 to 2X at 24-48 hours when drying off or while dry.

Coliform

Dihydrostreptomycin — ½-1 gm. + 200 T.U. penicillin in either aqueous or water-in-oil emulsion vehicle.[2]	1 to 3X at 12-24 hours if aqueous base, or 24-72 hours if emulsion is used. If septic, use every 12 hours.	1X when drying off. 1X when springing.

T.U. = thousand units.
[1] Penicillin G Procaine in aqueous suspension.
Penicillin G—Crystalline—not Procaine.
[2] Use vehicle with neutral pH for best results.
[3] Safe for use on acute cases.
[4] Too irritant for use on acute cases.
[5] Add penicillin to sulfas immediately before use.

Pseudomonas Types

Fifty mg. polymyxin B in 20 cc. of 20% aqueous solution of sodium iodide repeated 2 to 3 times at 24-72 hours has produced 40% or more recoveries in cases treated. Neomycin—0.5 gm. in water-in-oil emulsion or aqueous or aqueous solution gives approximately the same recovery. For known *Paracoli* a mixture of these two and 0.5 gm. dihydrostreptomycin is used.

Treatment often fails to remove infection but may improve clinical condition.

Acute Mastitis.—Infuse the affected quarter with at least 200 T.U. penicillin and at least ¼ to ½ gram of dihydrosterptomycin in 50 cc. of either sterile distilled water, saline, or an aqueous solution of sulfamerazine or sulfamethazine. This treatment is more effective when secretions are "let down" with 2-5 cc. posterior pituitary extract intravenously and all secretions removed. (Repeat before each infusion.)

The quarter may be packed with cracked ice until the hard tense swelling subsides. While the quarter is packed in ice, "let-down" is stimulated and secretion removed at 8-12 hour intervals and reinfused as described above. Under certain conditions bathing the quarter with cold water and stripping the secretion at 20-30 minute intervals materially helps to reduce the severe inflammation. When this practice is followed, the cow is systematically treated, but generally the udder is not infused until the frequent stripping has been stopped.

Systemic treatments are often indicated. An intramuscular injection of 1.5 to 3 or more million units of penicillin in oil and 1 to 5 grams of dihydrostreptomycin is given (the two can be mixed). Some practitioners use 1 to 5 grams of dihydrostreptomycin for the quarter infusion. The dihydrostreptomycin intramuscular injection should be repeated at about 6 hour intervals to maintain therapeutic levels. An aqueous solution of sulfonamides is frequently administered intraven-

ously at the rate of ¾ grain per pound of body weight, which is followed by oral sulfonamides (sulfathiazole or sulfamethazine or other sulfas) for two days. Caution must be used to prevent sulfonamide poisoning in those septic or toxic cows which may have reduced water intake. The use of blood transfusions and dextrose and saline solutions is occasionally important.

A few cases have apparently improved following repeated intravenous injections of 5 gm. daily of aureomycin or terramycin. Antihistimine therapy may be used.

Remarks.—*Intramuscular injections* of three million units of Procaine Penicillin and/or 1 to 5 gm. dihydrostreptomycin in stubborn clinical cases with known or unknown infections is helpful.

Sulfonamides in 10-25% solution per vein are useful in addition to infusion therapy in acute septic cases. (¾ gr. per lb. body weight.)

Treatment of hemolytic *Staphylococcus* and *Streptococcus* not *agalactiae* is usually recommended only when abnormal secretions are present or when there is a recent history of trouble in that quarter.

Records show one infusion of 100 T.U. penicillin in laboratory diagnosed *Streptococcus agalactiae,* clinically normal quarters, is highly successful, but in the treatment of clinical mastitis, no universal recommendations can be made to cover all cases. Good judgement must be used when deciding on the frequency and number of infusions. Favorable results can be obtained from one infusion in many cases, but two are better on grossly abnormal quarters. Larger amounts of antibiotics may be indicated where definite fibrosis exists. The sick (septic or toxic) cow may require more treatments depending on general condition and response to therapy.

Dry Cows.—If a cow has clinical flareups when being "dried off," she should be treated at that time. Infused quarters of dry cows should be closely watched for evidence of swelling or irritation for several days after treatment. If swelling or irritation does occur, milk out and handle as in lactating cows.

Owners should be cautioned to withhold secretion from treated quarters from market milk for 72 hours or until secretion becomes normal.

This list is not to be construed as meaning these are the only acceptable and efficient therapeutic procedures. They represent a summation of the common treatments which have consistently given good results in therapy of either laboratory diagnosed or clinical mastitis cases.

TREATMENT OF MASTITIS WITHOUT CAREFUL ASEPTIC TECHNIQUE BY THE VETERINARIAN AND ADEQUATE PREVENTIVE MEASURES BY THE DAIRYMAN HAS LITTLE PERMANENT VALUE.

For the veterinarian this means the use of sterile vehicles, syringes, and individual teat cannulae.

For the dairyman this means GOOD HERD MANAGEMENT with special emphasis placed on sanitation and proper use of a clean milking machine in good mechanical condition.

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PROTOZOAN DISEASES

Piroplasmosis

Piroplasmosis is a disease of the blood caused by various species of piroplasma, the generic name of a microorganism belonging to the order *Hemosporidia.* Three chief species of piroplasma are pathogenic to cattle: *P. bigeminum,* the cause of Texas fever in the United States, Australia, South America, parts of South Africa, the Balkan provinces, and tropical countries in general; *P. bovis,* the cause of enzootic hemoglobinuria in European cattle; and *P. parvum,* the cause of East African Coast fever. The piroplasms have been named in part after Babes (*Babesia*) and in part after Theiler (*Theileria*). Piroplasms are found in red blood cells, usually in the form of pear-shaped bodies arranged in pairs, though they may be in the form of circles or rods. These bodies contain nuclei and have an ameboid movement. Infection occurs through the bite of a tick. Reproduction apparently occurs in the blood or tissue cells of mammals, and the tick is an essential intermediate host. They destroy the red blood cells, causing anemia, hematogenous icterus, and hemoglobinuria.

TEXAS FEVER

(Tick Fever; Piroplasmosis; Splenic Fever; Red Water; Babesiasis)

Etiology.—*Prevalence.*—Texas fever first attracted attention in the United States in 1868, when cattle shipped from Texas caused heavy losses in Illinois and Indiana, where, as carriers, they introduced the piroplasma to the highly susceptible native cattle. Then followed the establishment of the Texas fever line, the boundary between the infected and noninfected areas. The geographical distribution is restricted to areas in which the cattle tick, *Boophilus annulatus (Margaropus annulatus)* may survive through the winter. The government program of tick eradication has reduced the infected area from sixteen states in 1906 to parts of two states in 1938—Florida and Texas. Immunity exists among native cattle in infected areas, and all cattle less than 15 months old are less susceptible than mature animals.

In animals less than one year old the reaction to infection may be so mild that no symptoms are observed. In the summer the disease is much more severe than in the winter, and premunition for shipment of susceptible animals to infected areas in Texas is practiced between November and March—Schmidt.[1]

Babesia bigemina, the cause of Texas fever, was discovered by Smith

and Kilborne[2] in 1889. In connection with their report, Salmon wrote "It has long been suspected by cattle owners that the appearance of the disease in northern cattle was in some way connected with the ticks distributed by southern cattle. This hypothesis has, however, been generally discredited by scientific men." In the report by Dr. Smith is this interesting paragraph: "During the summer of 1889 Dr. F. L. Kilborne, in arranging the various enclosures at the Experiment Station for the exposure of native cattle to the infection of Texas fever, conceived the happy idea of testing the popular theory of the relation of ticks to the disease. This he did by placing southern (North Carolina) cattle with native cattle in the same enclosure and picking the ticks from the southern stock as soon as they had grown large enough to be detected on the skin. This prevented any ticks from maturing and infecting the pasture with the eggs and hence prevented any ticks from infecting native cattle subsequently. At the same time, in another enclosure, the ticks were left on the southern cattle. The natives in the latter field died of Texas fever; those in the former did not show any signs of the disease." Among other conclusions Smith wrote that "southern cattle without ticks cannot infect a pasture," and "ticks alone scattered on a pasture will produce the disease." In 1893 Smith and Kilborne[3] published an extensive report in which they described the protozoan cause of Texas fever, creating for it the name *Pyrosoma bigeminum;* they also reported the production of Texas fever in cattle by the inoculation of blood from diseased animals.

In the body *Babesia (Piroplasma) bigemina* is present in the red blood cells during the febrile stage of the disease in the form of round or pear-shaped bodies from 2 to 4 microns long by 1 to 2 wide. The disease may be transmitted to susceptible cattle by intravenous injection of infected blood; equally effective are subcutaneous, intradermal, or intraperitoneal inoculations. The period of incubation is about one week. The blood of recovered animals is permanently infective. The ticks also carry the piroplasms; they attach themselves to the cattle, and infect them by means of numerous bites of the skin. The female tick falls to the ground and deposits from two to four thousand eggs. In from two to three weeks these develop into young ticks or larvae which climb the grass or weeds and finally gain access to the skins of cattle where they develop to maturity. The piroplasms are present in the eggs of the tick and through all stages of its growth. Susceptible cattle acquire the tick from tick-infested pastures, or from cattle originating in such pastures. In infected areas, native cattle are either immune or only slightly affected, and recovered animals are immune. Since the piroplasms remain in the blood of recovered cattle, these

animals may become a source of infection. Since the discovery of anaplasmosis in the United States, it has been recognized that in Texas fever *Anaplasma marginale* is also present.

Morbid Anatomy.—Postmortem decomposition is rapid. Ticks are attached to the skin between the thighs and upon the udder. Beneath the skin there may be paleness, yellowish color, and edema along the ventral surface of the body. On opening of the abdominal cavity, hyperemic spots may be observed on the omentum. The *spleen* is enlarged from two to four times the normal size. The blood is thin and watery. Mohler[4] writes that "probably the most marked pathologic alterations in the disease are found in the liver. This organ is very much enlarged and has a yellowish, mahogany-brown color, due to the bile it contains. This secretion becomes excessive, and minute plugs of congealed bile form in the small bile ducts, thus stopping them and damming the bile in the organ, which produces the yellow color. This does not occur evenly throughout the organ, and consequently it has a mottled appearance." The bladder contains urine that may or may not be bloodstained. No characteristic lesions are found in the stomach, intestines, heart, or the lungs.

Symptoms.—The incubation period is from one to two weeks, and all exposed susceptible individuals are taken sick at the same time. Two chief types are described: the acute form occurring in the hot summer or in highly susceptible cattle, and the chronic form occurring in partially immune southern cattle and in nonimmune cattle infected in the late fall.

In the *acute* form the onset is sudden, with depression, loss of appetite, and a temperature of 104° to 107° F. Fever may prevail for one or two days previous to other symptoms. The visible mucous membranes are often icteric, though in the chronic form they are pale. Constipation is always present at first, but it may be followed by diarrhea. The pulse and respirations are greatly increased in frequency. Hemoglobinuria is usually present, the color of the urine varying from light red to black according to the rate of destruction of the red blood cells. The blood is light colored and clots slowly. In severe types the red cell count drops from 7 to 8 millions to 1 or 2 millions, and on microscopic examination many piroplasma are found in the red cells. Death usually occurs within a week in mature susceptible animals in the hot summer months, the mortality being about 90 per cent. In animals under nine months of age the course is usually short and seldom fatal; the mortality in yearlings is about 25 per cent; from eighteen months to two years, about 50 per cent—Mohler.[4]

In from three to six weeks after recovery from an acute attack there

may be a relapse, in a mild or chronic form, with a second period of destruction of red blood cells. This second attack may possibly be due to concurrent infection with anaplasma.

The *chronic* form appears under natural conditions in Texas-fever areas in the late fall or early winter, and it may be produced experimentally by placing a few ticks on susceptible cattle.

The symptoms are like those of the acute form, except in degree. Usually there is no discoloration of the urine because of the slight or gradual destruction of red cells. The mucous membranes are pale, there is a gradual loss in condition, and the temperature is about 103° F. The course is irregular, the mortality low, and recovery is often incomplete. The febrile course may extend over weeks or months.

Control.—The presence of Texas fever in any cattle-raising country places serious restrictions upon the industry, because of persistent and permanent damage to the native cattle, and because of the high death rate among imported improved susceptible breeds. In the United States, infected areas in the South have rapidly been decreasing since 1906 under the tick-eradication program of the Government. As described by Graybill,[5] two chief methods of destruction of ticks are employed: (*a*) pasture rotation, in which all animals are removed from infested pastures until all the ticks have died of starvation—8 to 10 months; and (*b*) treating the animal with an agent that will destroy all the ticks present—crude petroleum, arsenical dips, toxaphene.

Premunition.—Artificial immunity may be established, by means of inoculation of young cattle with infected blood, which produces a mild form of the disease, or by the application of a few ticks, which also results in a mild form of Texas fever. Francis[6] was successful in the introduction of susceptible cattle into Texas by importing young calves, 4 to 6 weeks of age. The calves are immediately placed upon nurse cows, preferably in the winter months. Vaccination has not proved to be successful in animals more than one year of age. As described by Schmidt, "inoculations were practiced between November 1 and March 1, in rare exceptions later in March. In most cases the virulent blood was drawn from a tick-infested animal; in some cases from an animal that had been free of ticks for from one to five years or had never had ticks on it but had been inoculated at least one year previously with blood containing both anaplasmata and piroplasmata. The animals immunized all came from tick-free territory and from many states in the Union. On account of the wide spread of anaplasmosis as well as piroplasmosis in the vast area of Texas then infested by ticks, it was, of course, necessary to immunize cattle against both piroplasmosis and anaplasmosis." Inoculation with a combination of

the two is followed by two fever reactions; the first, due to piroplasms, beginning on the third to the sixteenth day; and the second, due to anaplasms, beginning on the seventeenth to the forty-eighth day. The febrile reaction lasts from one day to two weeks or more and varies in type and continuity.

Premunition against tick fever varies in different countries according to the variations in species of piroplasms and the presence of anaplasms. In Australia, for example, Legg[7] reports the presence of *Babesia bigemina* (large piroplasm), *Babesia* (Babesiella) *argentinum* (small piroplasm), and *Anaplasma marginale.* Carriers of *B. bigemina* are highly resistant to artificial incoulation with *B. argentinum,* but they may succumb to natural infection from a tick. Legg writes that "to cope with natural infection with *A. marginale, A. centrale* (S. Africa) was added to the vaccine and the two organisms (*P. bigeminum* and *A. centrale*) inoculated together. . . . Inoculation of the babesiella immediately after the subsidence of the piroplasm reaction appears to be a satisfactory method of dealing with stud stock." This method of immunization or premunization employs a technic introduced by Theiler and described by du Toit as follows: "Theiler[8] in 1912 found a strain of *Anaplasma* (*A. marginale* var. *centrale* or *A. centrale,* for short). . . . Theiler then introduced a system of immunization in which cattle were injected with a small quantity (5 cc.) of fresh blood from a reservoir animal chronically affected with *A. centrale.* These cattle then contract a mild form of anaplasmosis with *A. centrale* in the blood, and this gives them a protection against the more virulent *A. marginale.*"

Premunition against combined piroplasmosis and anaplasmosis is practiced in Brazil with blood from a carrier animal that has been kept tick-free for a year. Three doses of 10-cc. each are given at 15-20 day intervals. The first is stored for fifteen days in a refrigerator before use, the second dose for twelve days and the third dose consists of fresh blood—Dupont.[9]

Treatment.—Chemotherapy is effective in the treatment of tick fever caused by any of the piroplasms. Acaprin (Bayer) is reported by Legg to be very valuable in combating both piroplasmosis and babesiellosis, whether naturally or artificially acquired. According to Idnani[10] it will free the blood from *B. bigemina* within 48 hours. Trypaflavine (acriflavine) (0.5 to 1 gm.) 1:1000 per vein also exerts a curative action against all forms of Babesia.

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EAST AFRICAN COAST FEVER

(Rhodesia Fever; Rhodesian Red Water)

Etiology.—East Coast fever is a form of piroplasmosis in cattle which occurs chiefly in East and South Africa. It is caused by *Theileria parva (Piroplasma parvum)* the smallest of the known piroplasms, and it is probably responsible for a greater economic wastage than any other member of the group—Daubney.[1] Transmission is by various ticks: *Rhipicephalus appendiculatus* and others. Infection occurs mainly in cattle less than a year old at pasture when the grass is high and wet. Unlike Texas fever it cannot be transmitted by blood inoculation, since it does not multiply in the blood stream but in the endothelial cells of the lymph glands, liver, and spleen.

Symptoms.—The period of incubation is from 10 to 12 days. The onset is sudden and marked by high fever, salivation, bloody feces, and swelling of the superficial lymph glands. Emaciation and weakness soon follow. In the red blood corpuscles one finds many small piroplasma. Anemia and hemoglobinuria are usually absent, and the appetite may remain until near the end. The mortality is from 60 to 100 per cent. According to Purvis,[2] "one attack followed by an interval of protection and reexposure to infection, may be followed by another attack."

Prophylaxis by premunition, and *treatment* by chemotherapy have been a failure. *Suppression* has resulted in a decrease in the number of outbreaks in the Union of South Africa from 290 in 1921 to about 40 in 1936. De Kock[3] reports that "the Department has depended on one of three methods: slaughter, removal of the cattle by quarantine, and dipping . . . and it may be possible to eradicate this disease completely from the Union, provided new infection is not introduced from outside."

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PIROPLASMOSIS IN EUROPEAN CATTLE

(May Disease; Red Water; Moor Disease)

Etiology.—In many parts of Europe there exists a hemoglobinuria of cattle caused by *Piroplasma (Babesia) bovis;* it is chiefly transmitted by the tick *Ixodes ricinus.* Distribution is in native woody or swampy pastures, chiefly in the spring months. Formerly it was attributed to the eating of plants rich in turpentine. It occurs as an enzootic in pastured cattle and young stock, rarely as a sporadic stable disease when ticks are brought to the buildings on bedding and roughage. It prevails through the spring and early summer, and occasionally in the fall. The ticks are found on the grass and bushes in moist swampy places, where they become permanently established—"bloody urine farms." Cattle from noninfected farms are less resistant to the disease than native animals.

Symptoms.—The period of incubation is about ten days. The first symptoms are diarrhea and a high fever. After one or two days the urine becomes dark red or black, even tar-like, according to the rate of destruction of red blood cells. The mucous membranes are at first red, becoming icteric and anemic. Severe cases show marked depression, weakness, and edema of the skin. The blood is thin and watery. On microscopic examination of the red blood cells many piroplasma are found. The prognosis is said to be favorable if affected animals are taken to the stable in the early stages of the disease and the ticks removed. The course is about two weeks. If the disease becomes chronic, death may result from emaciation and weakness.

Treatment.—Infected herds are stabled and the ticks removed with baths of creolin, crude petroleum, etc. All animals are fed abundantly on roughage and grain. Trypan blue (1 gram intravenously) is claimed to be effective, particularly in the less severe forms. Acaprin and trypaflavine are reported to be more effective than trypan blue. Prevention consists in avoiding infected pastures, and cultivation of the soil. Cattle may be immunized by inoculation with blood of recovered animals, but this method is advised only where the annual loss is more than 1 per cent.

PIROPLASMOSIS OF EQUINES

(Equine Malaria; Babesiosis; Nuttalliosis; Biliary Fever)

Etiology.—Piroplasmosis in horses is prevalent in France, Italy, Macedonia, Russia, India, Africa, Egypt, Central America and South America. It is caused by the piroplasms *Babesia caballi*, and *Babesia (Nuttallia) equi*. It is transmitted by the bites of various ticks: *Dermacentor reticulatus* and *Hyalomma aegyptium* in Europe, and *Rhipicephalus evertsi* in Africa. In addition to the horse, the mule, ass, and zebra are susceptible. It may be transmitted experimentally with the blood of immune animals, and horses brought to permanently infected countries are particularly susceptible.

Symptoms.—The period of incubation is from one to three weeks. The symptoms are remittent fever, severe heart weakness with a marked increase in the frequency of the pulse, icterus, and hemorrhages in the conjunctival mucosa. Other symptoms are marked depression, dyspnea, early constipation followed by diarrhea, subcutaneous edema, emaciation, polyuria, and dark yellowish discoloration of the urine. In acute cases, piroplasma are found in 50 per cent of the red blood corpuscles, and the red cells drop to 3 million. The course varies widely; death may occur within a week, or only after weeks or months. The mortality is high among horses that have not previously been exposed. Thus Basset[1] and Auger report 100 per cent mortality in untreated horses in southeastern France, following the importation of infected animals. Recovered animals are usually immune. *Autopsy* shows marked enlargement of the spleen. Often the lymph glands are hemorrhagic and swollen. Extensive reports on this disease in South Africa have been made by Theiler.[2]

Treatment.—Excellent results are reported from the action of trypaflavine (1 gm.) administered intravenously in 1000 cc. water, and from acaprin (1.2 cc. of a 5 per cent solution per 100 kg. body weight). Weisman[3] has reported that in Panama best results were obtained from neoarsphenamine (3 gm. in 30 cc. distilled water) once a week, intravenously.

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ANAPLASMOSIS OF CATTLE

(South African Gall Sickness)

Etiology.—In discussing the mild, chronic type of Texas fever, Moore[1] mentioned a spherical or coccus form described by Smith and Kilborne[2] as a different species of *Piroplasma bigeminum*, thought to be the cause of the mild form of Texas fever. He also referred to the opinion of Theiler[3] that this is not a form of *P. bigeminum*, but a new genus—*Anaplasma*. Any doubt concerning the relation of these associated protozoan forms has been dispelled by the the discovery of anaplasmosis over wide areas of the United States, and in sections where Texas fever has never been recognized, such as Montana and Wyoming, and 25 other states; but the major enzootic anaplasmosis is in the original Texas fever states.[6] In a report in 1928 Boynton[4] states that the disease was first suspected in California in 1925, and that in 1927 the Chief of the Bureau of Animal Industry, Dr. Mohler, had reported the presence of the disease in Kansas and Louisiana. Apparently it has existed in the United States as long as Texas fever. Stiles[5] has reported its presence in 21 states and believes that it may be expected in the northern states; heavy losses have been reported from Oklahoma. In 1949 anaplasmosis was brought to Minnesota in a shipment of Hereford cattle from several western states.[21] The disease is widely distributed in warm climates, and it has been described in various parts of South America, Africa, Asia, and Southern Europe. Its presence in Kansas and Missouri indicates that it is adapted to cool climates. Similar to Texas fever, anaplasmosis attacks mature cattle. Calves under one year of age are rarely affected, and only in a mild form. It is most prevalent in the warm months, beginning in July and ending in October or November. Recovered animals are permanently immune. There is a notable difference in virulence between blood from different carrier animals.[6]

The Infection.—*Anaplasma marginale* was first identified as an independent parasite by Theiler.[3] Observing that these organisms had lost their body plasma and were transmitted only in blood containing corpuscles, he named them *Anaplasma*. They are round or oval intercellular organisms having a diameter of from 0.1 to 0.6 microns. *Within the body* they are found only in red blood cells, as high as 50 per cent of which may be infected during the febrile stage of the disease; they are also present indefinitely in the blood of recovered cattle.

Outside the body the habitat is not entirely known. Boynton reported that in California in counties where the disease was very prevalent the cattle on many of the infected farms were free from ticks; large horse flies were suspected to be the carriers. In 1941 Lotze and Yiengst[20] re-

ported experimental transmission of anaplasmosis by the horsefly *(Tabanus)*, and in the same year it was experimentally transmitted by mosquitoes.[16] In localities where Texas fever prevails, both piroplasma and anaplasma are carried by a common tick—*Boophilus annulatus*. In general, ticks are carriers of anaplasma, and according to Boynton[7] 19 species have been proved vectors. In 1933 Stiles[8] reported that four different species of horseflies had been found to be capable of transmitting anaplasmosis in the United States. Dickmans[9] has reported experimental evidence incriminating 15 different species of ticks, 10 species of flies, and three species of mosquitoes as capable vectors of anaplasmosis in various parts of the world. *Tabanidae* (horse flies) are the most important natural vectors.[6] The disease has been transmitted repeatedly by dehorning operations. This suggests the necessity of thorough disinfection of needles and other surgical instruments after drawing blood or operating upon a bovine in infected areas. Additional study is required to determine the mode of conveyance of this disease in the United States. According to Boynton,[10] "the history of most outbreaks in California could probably be traced to mechanical transmission by human agents who failed to observe aseptic precautions in the dehorning, castrating, ear-marking and other operations of cattle herds which may include one or more carriers. There is strong evidence that cases of anaplasmosis have followed administration of anthrax, hemorrhagic septicemia, and blackleg vaccines and aggressins in which the same needle and syringe were used for inoculating an entire herd that happened to include some carriers." Several outbreaks were definitely traced to the bleeding of cattle for the abortion test. Other possible carriers are nose-tongs, pitchforks, a stick with a nail in the end, or any sharp device employed for driving cattle to their stanchions.

Morbid Anatomy.—All visible mucous membranes and the skin are anemic and icteric, and there may be edema of the subcutis along the ventral surface of the neck and chest. The spleen is enlarged and degenerated. The liver is swollen and icteric. The gall bladder is distended with thick greenish brown bile. Icterus is general and marked throughout all the organs of the body, including the central nervous system. In the chronic form the liver is about twice the normal size and badly degenerated, the blood is watery, and the bone marrow yellow. The lungs are anemic or icteric, sometimes emphysematous, and petechiae may be found on the pleura. The kidneys are normal, and only slight changes are found in the digestive tract.

Symptoms.—After experimental inoculation of blood the period of incubation is from twenty to forty days. Boynton[10] describes four types of the disease: mild, peracute, acute, and chronic.

The *mild* type occurs usually in calves. There are slight general symptoms, such as rough coat, dullness, lack of appetite, slight constipation, and loss of condition. A few show a whitish mucopurulent discharge from the nostrils and eyes. Recovery occurs in a few days. Marginal bodies are present in the red blood cells.

The *peracute* form prevails chiefly in milch cows. The onset is sudden with marked depression, complete suspension of the milk flow, and a high fever. The ears droop, the muzzle is dry, and saliva drools from the lips. Death occurs within a few hours. Diagnosis depends on finding marginal bodies in the red blood cells; as high as 50 to 75 per cent may be infected.

The *acute* type presents marked general weakness with a temperature above 104° F. Boynton[10] mentions accelerated and labored respiration, a pulse of 80 to 100, a pallid or slightly icteric conjunctival mucosa, and in some cases a mucoid nasal discharge. Muscular tremors are often present. As the course approaches a fatal end, the skin and all visible membranes become anemic and jaundiced. Urination is frequent but the urine is not bloody as in Texas fever. Constipation and paralysis of the rumen are the rule, and abortion is frequent. The mortality is high, death occurring after two or three days. In recovered cases the convalescence is slow. Convalescents show a desire to eat dirt.

Boynton[10] writes that after the blood cells have dropped to three or four millions, and the hemoglobin to 40, regeneration may lead to swift recovery within a few weeks, provided it begins before jaundice appears. In 175 field cases Smith and Howell[11] observed a high mortality in animals with less than 30 per cent hemoglobin (Tallquist), while those showing more than 50 per cent usually recovered. Regeneration is first detected by the appearance of megalocytes in the blood. If regeneration does not occur, the red cell count may drop to one million and the hemoglobin to 15 per cent. The corresponding symptoms are a pulse rate of 150 to 170, thirst, jaundice, prostration, muscular tremors, and salivary drooling.

The *chronic* form is merely a continuation of a severe acute attack in which the red cells drop to approximately one or two millions. With the appearance of young red cells in the blood, the marginal bodies rapidly decrease. The clinical symptoms are a continuous low-grade fever, anorexia, thirst, emaciation, high pulse, marked jaundice, and occasionally hemoglobinuria. Three or four months are required for complete recovery. Extreme anemia may lead to intense generalized icterus and death.

The mortality varies widely in different herds, the average is given at from 30 to 50 per cent.

Diagnosis.—In Texas-fever districts it has obviously been many

times associated with that disease. Outside these areas it may be mistaken for anthrax, hemorrhagic septicemia, blackleg, and poisoning. While the symptoms, lesions, and epidemiology are sufficiently distinctive to identify anaplasmosis in the majority of cases, a microscopic examination of the blood will disclose the characteristic marginal bodies, for a brief period, since *Anaplasmata* usually are not detected in the blood after recovery.[6] Mohler *et al.*[17] have reported promising results, using a complement fixation procedure which they modified from a similar technique used in diagnosing malaria. In severe acute general infections, congestion and hemorrhage of the serous and mucous membranes are in marked contrast to the anemia and jaundice observed in anaplasmosis.

In a Federal Report[12] it is stated that "calves from 1 to 3 months of age become very susceptible to anaplasmosis following splenectomy, with marked symptoms of fever and anemia and with a high percentage of their erythrocytes containing marginal bodies. . . . These young splenectomized calves have proved to be very economical and reliable test animals for anaplasmosis."

Treatment.—Sick animals should be kept quiet and not forced to move about. Provide plenty of food, free access to water, and protect against flies. Weakness may be combatted with stimulants of strychnine, camphor, or caffeine. Avoid laxatives and drugs that may weaken the animal. Trypan blue has proved of no value. It is doubtful if any medicinal treatment exerts a favorable action on the course of the disease. Boynton[13] and associates have reported curative action from dextrose (one liter of a 5 per cent solution, to which has been added sodium cacodylate (25-30 grains per 100 lbs. body weight); this is administered intravenously. Schmidt[14] writes that "at the present time there is no drug known that gives satisfactory results in the treatment of anaplasmosis." In the treatments reported by Smith and Howell[15], neosalvarsan, tryparsamide, cobalt chloride, and sodium sulfathiazole, each in combination with large amounts of dextrose, gave the most promising results.

Prophylaxis.—In infected areas in the United States care must be taken to avoid accidental inoculation by means of infected instruments and syringes. In the use of hypodermic needles for the administration of biologics or drugs, or for drawing blood, a sterile needle should be used for each operation.

Preventive Inoculation against Piro- and Anaplasmosis.—Successful vaccination against this group of diseases has been developed chiefly for protection against piroplasmosis in purebred stock before its release

in a tick-infected country. Two methods are practiced: (1) Introduction of susceptible animals into infected areas in the winter when ticks are inactive. The disease is naturally contracted in a mild form and permanent immunity is conferred. (2) Animals having blood that conveys the disease in a mild form are kept free of ticks and their blood is used for artificial inoculation. Different strains of the disease exist, and it is important to inoculate with blood from an animal where the cattle are to be shipped. As practiced in South Africa, blood of known virulence is obtained from veterinary laboratories and used within forty-eight hours after being drawn, if the reaction is unduly severe the animal is treated with trypan blue (Edmonds).[18] Legg[19] has reported on the difficulties involved in immunization of stud stock where both anaplasma and piroplasma are prevalent. Schmidt[14] has reported that "the mortality from anaplasmosis among more than a thousand animals immunized at the age of 15 months or under was 1.3 per cent. In older animals, however, heavier losses may be expected. Thus of 19 two-year-old bulls inoculated with a pure strain of anaplasma, four died from anaplasmosis."

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TRYPANOSOMIASIS

The trypanosomes are a group of motile protozoa occurring in the blood plasma and other body fluids. They are from 20 to 25 microns long by 1.5 to 3 wide, and multiply by cell division. Hornby[1] states that at least twelve well-defined diseases of domestic animals are known to be due to various species of trypanosomes. With the exception of dourine, infection takes place through the bites of flies. The most important pathogenic trypanosomes and trypanosomic diseases are: *Trypanosoma equiperdum*, dourine of the horse; *Tr. brucei*, nagana or tsetse-fly disease; *Tr. evansi*, surra; *Tr. equinum*, mal de caderas in South America; *Tr. gambiense*, sleeping sickness in man.

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DOURINE

Definition.—Dourine is a contagious disease of solipeds transmitted by coitus. It is characterized primarily by inflammation of the external genital organs, secondarily by paralysis and a peculiar skin eruption caused by lesions in the peripheral nerves and the intervertebral ganglia. The infective agent is *Trypanosoma equiperdum*.

Etiology.—Dourine belongs to the group of animal plagues that have largely disappeared under the influence of modern hygiene. Its history in the United States dates from the reported entry of a Percheron stallion from France in 1882, and spread of the disease in Illinois until recognized by Williams[1] in 1886. In addition to the original outbreak

in Illinois, enzootics of the disease have been overcome in Nebraska, Iowa, North Dakota, South Dakota, Arizona, New Mexico and Wyoming. Mohler[2] wrote in 1920 that its complete eradication in the near future was expected. If the disease is in existence at the present time it is limited to the wild or semi-wild horses of the West. During the World War it entered Germany from Russia and in 1921 was reported from 237 farms in Germany; ten years later it had been entirely eradicated from this country. It is prevalent in Russia, Roumania, Algeria, Spain, India, and various parts of Asia.

Trypanosoma equiperdum.—While the blood of the horse is known to be the habitat of this trypanosome, it is rarely found there. It may be found in the fluid contents of recent swellings on the skin (dollar plaques), in the mucous discharge from the urethra and vagina, as well as in fluid obtained by puncture of the testicle. During copulation infection occurs from the secretions of the urethra and vaginal mucosa of diseased stallions and mares, the trypanosome being able to penetrate uninjured mucous membrane. Experimental inoculation may be accomplished in horses, dogs, cats, sheep, white mice, rats and rabbits. Intraperitoneal inoculation of mice causes death from septicemia in two to five days. Dogs succumb after two to three months; they show a characteristic ulcerative keratitis and exudative iritis.

Symptoms.—In experimental inoculation of the horse the period of incubation is from one to four weeks and it may be much longer.

Two stages of the disease are recognized: (1) The primary stage which corresponds to local infection of the genitals, and (2) secondary general infection with localization in the skin, nervous symptoms, and emaciation. The primary symptoms in stallions are edematous swelling of the prepuce, penis, and testicles, redness and swelling of the urethral mucosa, a slight mucopurulent discharge, and swelling of the inguinal lymph glands. In the mare the initial symptoms are edematous swelling of the vulva, redness and swelling of the vaginal mucosa, and a mucopurulent vaginal discharge. Increased sexual excitement is observed in both sexes. According to Williams[3] the vesicles and pustules mentioned by European writers are never observed as a symptom of dourine, and are not the result of any form of trypanosomiasis.

The secondary stage begins with the formation of urticaria-like plaques on the skin—dollar plaques, *Talerflecken*. These are firm, round, flat swellings, one to two inches in diameter, located on any part of the body or neck. They are pathognomonic of dourine, and may come and go in series. Depigmentation of the skin of the external genitals may also occur. The nervous symptoms consist of motor paralysis arising from peripheral neuritis; they consist of facial paralysis, sacral

paralysis with knuckling of the joints of the hind limbs, dragging of the feet, and abduction of the limbs. Paralysis and prolapse of the penis are frequent. Emaciation is a characteristic secondary symptom; usually this begins in the gluteal muscles and extends rapidly. Also, there may be swelling of the submaxillary lymph glands, nasal catarrh, abortion, and an inconstant fever.

The *course* is chronic in cool climates, and acute in the tropics. In the United States the symptoms may alternately improve and reappear over a period of months and years. Apparent recoveries are usually incomplete. The mortality is estimated at from 50 to 75 per cent. A positive *diagnosis*, even of latent cases, can be made by means of a complement-fixation examination of the blood. In the United States, where only one form of trypanosome prevails, this method of detection has led to probable extermination of the infection.

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NAGANA

(Tsetse-Fly Disease)

Nagana is an African trypanosomiasis caused by *Tr. brucei*. It is transmitted by the bite of the tsetse-fly *Glossina morsitans;* according to Hornby, it may also be transmitted mechanically, in the absence of this fly, especially to cattle and pigs. This author further states that "the number of domestic animals (other than poultry) found within tsetse 'belts' is comparatively insignificant. . . . All game or other large animals are resistant." The area of infection in Central and South Africa has been greatly restricted in recent years. Its chief prevalence is in horses and cattle, to a lesser degree in sheep, goats, swine, and dogs. The incubation period is from two to ten days. At first there is a high fever, marked congestion of the mucous membrane, and edematous swellings of the limbs, abdomen, eyelids, and submaxillary region. The course is chronic over a period of one to several months, during which there are irregular fever, anemia, icterus, marked weakness, and in some cases urticaria. Many trypanosomes are present in the blood. Recovery is infrequent. In 1941 Hornby[2] concluded that no method of immunization has yet been devised that will enable East African domestic stock to thrive in tsetse-fly belts.

Treatment consists in the intravenous injection of Bayer 205. The use of DDT and benzine-hexachloride sprayed from airplanes upon the dense bush which forms the main breeding places of the insects has reduced the tsetse-fly incidence to a level which may result in total elimination of the parsite.

SURRA

Surra is prevalent in India and other Asiatic countries, including the Philippines. It is caused by *Tr. evansi,* and is transmitted by various flies—*Tabanus, Stomoxys.* Its chief prevalence is in the horse, mule, ass, camel, elephant, and dog. Cattle and other animals that are only slightly susceptible may carry the virus. The incubation period is from seven to thirteen days. At first there is a high fever, anemia of the mucous membranes, edematous swellings of the skin, and urticaria. The course is chronic over a period of one to two months, during which there are emaciation, ataxia, sopor, irregular fever, icterus, and marked weakness. In general the symptoms are like those of nagana, and the trypanosomes are abundant in the blood in the early stages of the disease, as in nagana. Untreated horses usually die, while cattle usually recover. Of the *diagnosis,* Hornby states, "The formolgel test can be used with some success with equine serum; for camels it is almost specific, but has recently been superseded by the equally specific and more delicate mercuric-chloride one. This was devised by Bennett and Kenny; it consists in adding one drop of serum to 1 cc. of 1:25,000 mercuric-chloride solution, and watching for the opacity which signifies infection."

Treatment.—Hornby[1] reports that "in the case of animals having a high natural resistance curative treatment is easy; a single dose of any trypocidal drug being generally sufficient to cure buffaloes, sheep, and goats. Camels are also favorable subjects. The highly susceptible animals, particularly dogs and horses, are more difficult to treat. Best results are obtained from Bayer 205, alone or in conjunction with another drug, such as tartar emetic, antimosan, sulpharsenol, etc. . . ." In those countries in which the disease is widespread, *prophylaxis* generally takes the form of measures to protect the class of animal most affected. Thus in the Anglo-Egyptian Sudan military camels are adequately protected by regular application of mercuric-chloride test, and segregation and treatment of all reactors. In Java the presence of buffalo carriers renders eradication quite impossible, so Bakker dealt with outbreaks among horses in the following way: "When surra appeared, all horses in the district were injected with 1 gm. of Bayer 205, and horses from outside the district were not admitted unless also injected.

The animals could be used, so that ordinary business hardly suffered, and they were protected from infection for a month. Injections were repeated every month for two or three months, until the outbreak was suppressed. In South America, too, emphasis has been laid on the hopelessness of eradication, and the necessity of making the utmost of chemo-therapy."

According to Bubberman[3] treatment may be effective if given at the beginning of the attack. He recommends simultaneous injection of 3 to 3½ Gm. naganol and 3 to 3½ Gm. atoxyl per 150 to 200 kg. of body weight. Following this treatment the percentage of recoveries is said to be about 60 per cent. Yutuc[4] states that in the Philippines naganol-atoxyl therapy was found valueless in the treatment of equine surra. When naganol was combined with sodium antimony tartrate and administered in nonlethal but slightly toxic doses, 2 out of 5 artificially infected surra horses and 1 out of 3 naturally infected animals recovered from the disease. Furthermore from the field trial carried out in British North Borneo during the summer of 1938, of the 100 horses naturally infected and actually given the naganol-sodium antimony tartrate combination, 63 were cured of surra.

Mal de caderas is a South American trypanosomiasis occuring in Brazil, Argentina, and Paraguay. It is caused by *Tr. equinum* and affects chiefly solipeds. The natural method of infection is unknown. The symptoms are progressive emaciation, weakness of the hind quarters, and staggering gait. The symptoms and lesions are similar to those of nagana and surra.

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MURRINA

Murrina is a South American trypanosomiasis which closely resembles surra. It occurs in the Canal Zone, Panama, and probably in other Central American countries. It is caused by *Trypanosoma hippicum* that cannot be distinguished from *Tr. evansi,* the cause of surra. It is transmitted by flies and the vampire bat (*Desmodus rotundus*).

Cattle are not affected but they may carry the trypanosome in their blood. The period of incubation is five days and the disease begins with depression, weakness, and fever. Edema and fever are recurrent throughout the course, hemoglobinuria is frequent, and death occurs in from two to three months. A similar, and perhaps identical, disease occurs in Venezuela.

METAZOAN INFECTIONS

TRICHINOSIS

Trichina spiralis occurs in swine in two stages of development: sexually mature intestinal trichinae, and larval trichinae which are encapsulated in the muscles. The adult parasite is a round worm from 1.5 to 4 mm. in length with a pointed head and a somewhat rounded tail. Its habitat is the small intestine of hogs, rats, mice, and other mammals, including man. The larval form is from 0.6 to 1 mm. in length. When flesh containing the larvae is eaten by man or any animal in which development is possible, the capsule is digested and the trichinae are set free. They become sexually mature in the small intestine about the third day, and beginning with the seventh day the females deposit living embryos directly into the crypts of the intestinal glands. These embryos are carried in the circulation to the muscles where encapsulation begins in about four weeks and continues for three months. Calcification of the encapsuled trichinae begins from the third to the sixth month and continues for eighteen months; this change renders the cyst visible. The favorite locations of the larvae are in the muscular portions of the diaphragm, the pharynx, and the tongue, and to a lesser extent in the intercostal and abdominal muscles.

In *swine* natural infection does not induce symptoms. But after a heavy artificial infection there may be high fever, diarrhea, stiffness, colic, difficulty in eating and swallowing, dyspnea, and edema. Swine and rats are infected by eating scraps of raw pork or rats or mice containing the encysted parasites. But the chief source of infection is swine, and little importance is now attached to rodents as a source of infection. Schwartz[1] has reported that an examination of 6,662 samples of diaphragm muscle tissue obtained from 1933 to 1937 originating from grain-fed hogs revealed an infection of 0.91 per cent, as compared with an infection of 4.41 per cent in hogs fed on uncooked garbage. In a Federal[2] report for 1938 data are presented which show "ten times as great an incidence of trichinae in hogs fed garbage as in hogs not fed garbage. They also show that the intensity of infestation with trichinae in garbage-fed hogs is much greater than in grain-fed hogs." It is believed that the feeding of garbage to hogs is the main source of trichina infection. Skin tests for the detection of this disease in live swine are not sufficiently accurate (U.S.B.A.I.).

In *man* trichinosis may be a severe and fatal disease. The usual source of infection is from the ingestion of raw or insufficiently cooked

trichinous pork. The inspection of export pork for the detection of such infection was one of the chief reasons for the establishment of the Federal Bureau of Animal Industry. It is claimed that no case of trichinosis has ever been traced to the consumption of meat exported from the United States. Sawitz[3] reports that the number of clinical cases of trichinosis reported to the United States Public Health Service for the years 1915-36 is 2,968, and that the yearly increase in incidence is partly explained by an increased interest in the disease. The mortality is 4.4 per cent. "The greater prevalence of the disease in the Midwest and other areas where Germans and Italians have settled reflects local customs in the preparation of food. . . . Autopsy studies indicate that approximately 16 per cent of the population is infected."[4]

Destruction of trichina in pork is accomplished by thorough curing, by refrigeration for a continuous period of not less than 20 days at a temperature of not higher than 5° F., and by heating to 131° F.—Schwartz.

Prevention consists in the thorough cooking of all pork before it is eaten, either by humans or swine.

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TAENIASIS

Taenia saginata (Beef Tapeworm) is the most common tapeworm of man in North America. The mature parasite is harbored in the small intestine of man. It is a flat, white, jointed worm, from 12 to 25 feet or more in length, and usually only a single worm is present. Cattle become infected with the larval form (*Cysticercus bovis*) by swallowing eggs in food or water contaminated with infected human feces. The larvae penetrate the intestinal wall and develop into the cysticerci in the flesh or organs. The favorite locations are in the masseter muscles and heart. The presence of these cysts constitutes the condition known as measles ("measly beef"). There are no clinical symptoms in animals. Man acquires the infection by eating raw or insufficiently cooked meat infected with cysticerci.

Taenia solium (Pork Tapeworm) is not common in the United States. It is said to be somewhat more frequent in Continental Europe and Asia. The adult worm is 6 to 12 feet in length. Hogs become infected

with the larval form by swallowing food or water contaminated with eggs from human sources. The larvae pass to the muscles and organs and become encysted (*Cysticercus cellulosae*). The cysts are oval, whitish bodies from 1/8 to 1/4 inch in diameter. Man acquires the infection by eating raw or insufficiently cooked pork infected with cysticerci ("measly pork"). Both the larvae and the adult worm may develop in man.

The symptoms of taeniasis in man are due to the presence of the parasite in the intestine, and frequently they are severe. Since man may also become infected in the muscles with the larval form of *T. solium,* this parasite may cause even more serious symptoms.

DISEASES OF ALLERGY

ANAPHYLAXIS

(Hypersensitiveness; Sensitization; Idiosyncrasy; Atopy)

Definition.—Allergy is a reaction which occurs when an antigen is administered parenterally to an individual whose cells have been sensitized to this specific antigen.

Etiology.—Sensitization of the cells, the allergic state or condition, is accomplished by contact with a wide variety of antigens, such as bacteria and their products, pollen, sera, drugs, foods, oils, resins, and many others. It has been thought that proteins are the only substances capable of bringing about the anaphylactic state, and in large animals they are the causes of sensitization in the vast majority of cases. Furthermore, these proteins must be foreign to the animal receiving them. Antigens may be absorbed by ingestion, inhalation, injection, or from a focus of infection. In the process of sensitization by contact, antibodies are formed in the cells, and thereafter the parenteral injection of the antigen results in an altered linkage of the cells to the antigen with an explosive reaction called anaphylactic shock. This is the usual form in animals and it occurs immediately or shortly after injection. Such "serum accidents" are regarded by most authorities as examples of anaphylaxis. This artificial sensitiveness was reported by Theobald Smith in 1904, who observed that many of his experimental animals died immediately after a second injection of serum after a sufficient interval of time. It is the sensitized condition of the animal which results in an anaphylactic shock; the antigen itself is harmless to normal individuals. The phenomena of allergy are clothed in mystery; nobody knows what an antibody is or any of the details of the explosive reaction.

In most cases it is impossible to prove contact with the antigen prior to sensitization; yet it is impossible to prove the entire absence of such contact, which may have been direct, or intra-uterine, or inherited. Perplexity over a loss to explain the occurrence of anaphylaxis in animals that are injected for the first time has been expressed by Reichel,[1] who assumes that inasmuch as the anaphylaxis is observed these animals are "naturally sensitive" to the foreign protein. Mackenzie[2] refers to first injection serum accidents in man as occurring in "spontaneously hypersensitive individuals." In both man and animals the majority of serum accidents follow first injections. Another explanation may be found in the theory that the shock following the injection of serum is

not anaphylactic, but is caused by some unknown toxic substance in the serum itself. In June 1943 there was available a current series of anti-hemorrhagic-septicemia serum derived from the blood of cattle that was rapidly fatal to cattle when the prescribed dose was injected either subcutaneously or intravenously. One 2-year-old heifer died following a subcutaneous test injection of 5 cc., and extensive pulmonary edema was found on autopsy; a young calf died following a subcutaneous injection of 40 cc., and pulmonary edema with pulmonary hemorrhage was found on autopsy; another young calf collapsed after receiving 15 cc., intravenously, but finally recovered; another young calf died within 10 minutes after receiving 50 cc., intravenously. So far as known this series of serum caused either reaction or death in every animal injected, regardless of the dose or the manner of administration. In such cases it is illogical to assume that "the antigen itself is harmless."

Pasteurization of homologous serum at 58° or 59°C. for 30 minutes, with the subsequent addition of phenolic preservative, causes alterations or changes that are more apt to be followed by the occurrence of anaphylaxis than occurs in the use of the same serum not so altered by the heating process (Reichel). Pasteurization is extensively employed for the purpose of controlling virus infections. The observation is recorded by Reichel that since the aging of serum with phenolic preservatives over a 30-day period will effectively destroy the infectivity of serum, including the virus of swamp fever, it is more than likely that other infections will succumb in a similar manner. Severe reactions and losses in horses following the second injection of chick-embryo equine encephalomyelitis vaccine have been reported by Schoening,[3] who suggested "a possibility that through the aging of the vaccine there might have developed certain substances highly antigenic (but not necessarily related to the specific virus) so that when some animals that were particularly sensitive to foreign protein were injected with the second dose of the same product, severe reactions and death might occur." These reactions have been avoided with the adoption of the intradermic administration of chick-embryo vaccine.

In the selection of sera or aggressins, or biological products containing sera, they should be derived whenever possible from animals of the same species as those to be injected (homologous). In the use of anti-hemorrhagic-septicemia products in cattle, for example, those of equine origin should be avoided. Fincher,[4] and Fincher and Gibbons[5] have reported that "urticaria-like swellings have been seen in our cases only when a serum or aggressin had been used that was of equine origin." Anaphylaxis following the use of bacterins is not rare, and the reactions may be severe or even fatal. Such shocks may be due to

horse serum used to fortify broth cultures or agar media. To avoid anaphylaxis, bacterins should preferably be an actual bacterial suspension in a dilution of physiological solution free from toxic broth or other foreign protein.

Bacterins and vaccines are in our experience the most frequent causes of anaphylactic shock. There have been numerous reports of fatal reactions in calves following the administration of bacterins for the prevention or treatment of scours. In one instance two 6- to 8-month-old calves received 5 cc. hemorrhagic septicemia bacterin as a prophylactic against possible exposure. About three hours later they were found in extreme dyspnea, and breathing with open mouths in short gasps that could be heard for at least 30 feet. Death occurred about ten minutes later, and extensive edema and hyperemia of the lungs were found on autopsy. In the routine annual vaccination of a herd with bacterins against hemorrhagic septicemia before travel on the show circuit, one valuable cow was found dead within an hour after the injection; apparently she had dropped in the stall and died without a struggle. The use of calf scour serum, or the dam's blood, may cause severe reactions in young calves. Other examples are blood transfusion in mature animals; injection of a sow with a prophylactic dose of anti-swine-erysipelas serum (fatal); and injection of a cow with pregnant mare serum (Gonadin), resulting in urticaria. It is possible that the occasional fatal attack of edema of the lungs in cows of unknown cause is anaphylaxis. Other probable examples of anaphylaxis in cows are urticaria apparently caused by larvae of warble flies, and "summer snuffles" of unknown cause. Shock sometimes follows intravenous injection of calcium gluconate, neoarsphenamine and formalin, the reaction is not usually severe, but such accidents have terminated fatally.

Symptoms.—Following the injection of a hemorrhagic septicemia bacterin there may be dyspnea, and trembling. The onset may occur immediately, or within an hour. Dyspnea and symptoms of pulmonary emphysema may persist for twenty-four hours. Urticaria-like symptoms are frequent in the form of edematous swellings around the muzzle, the eyes, and over all parts of the body. These are accompanied by intense itching, symptoms of colic, and extreme dyspnea with rapid breathing. Such swellings may develop almost immediately or after an interval of twenty-four hours. In severe attacks there is open mouth breathing with symptoms of edema and emphysema of the lungs that may persist for several days. There may be collapse immediately after the injection with recovery, or death within a few minutes. Other symptoms are lachrymation, coughing, and pulmonary hemorrhage with

bloody froth at the nostrils. The postmortem lesions consist of pulmonary edema and emphysema, or there may be absence of lesions with the exception of a few petechial hemorrhages.

Treatment.—Epinephrine (adrenalin 1:1000 solution) in doses of 3 to 8 cc. administered subcutaneously is a specific for serum accidents, and it should be available for immediate use whenever bacterins or serums are used. Injection may be repeated after a few minutes if necessary. As a prophylactic adrenalin may be added to the serum before injection. Sensitized animals may be recognized by the use of a test dose of serum employed by dropping a 1:10 dilution of the serum into the eye of the animal or the injection of such solution intradermally for the purpose of determining the local anaphylactic reaction. Another method is to inject 5 cc. of the serum subcutaneously to determine sensitiveness prior to injection of the prophylactic or therapeutic dose.

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POISONING

"Suspected poisoning" from plants, spoiled food, and criminal or careless use of poisons is a frequent provisional diagnosis when animals are sick from an unknown cause. Accidental and criminal poisoning cause many deaths in animals, and in suspected cases, where it is important to make a positive diagnosis and to place the responsibility, the veterinarian must be familiar with both the legal and the pathological aspects of the case. If criminal poisoning is suspected, the veterinarian should ask that a responsible public official be present at the investigation of the case or premises, in order to take personal charge of any materials or tissues that may be used as evidence. This guards against a defense that the plaintiff, or some person employed by him, might have added poison to the material when in transit to the chemist.

Food poisoning is important in relation to the introduction of food or supplemental food substances not previously used. Examples are the introduction of sweet clover, vetch, and India peas, the contamination of roughage with poisonous plants, such as equisetum or lupines, the use of rock phosphate or phosphatic limestone as a mineral supplement, and the use of food spoiled by decomposition in which the toxic agent may be detected (botulism), but more often merely suspected.

There is a tendency to attribute the cause of every unknown disease to "some form of poisoning." For this reason it is important for a veterinarian to be familiar with the most probable toxic agents in his locality. In many sections of the eastern United States these are metallic poisons; in many parts of the West, plant poisons are more important. Overstocking of dry pastures or ranges often leads to the eating of plants not commonly consumed, such as lupines, senechio, and brakes. Semistarvation under any circumstances brings its own peculiar trail of disease.

Requirements and whims of modern agriculture have brought to the farm many toxic substances formerly unknown to the dairy farmer. Examples are nitrate of soda for fertilizer, arsenate of lead for destroying plant pests, calcium cyanide for poisoning woodchucks, other cyanide preparations for insecticides, and cresol and other poisonous substances under a myriad of trade names. Familiarity with these substances breeds carelessness and affords an easy opportunity to the criminal and halfwit. Among the dangerous therapeutic agents recommended in popular literature and easily obtained are carbon tetrachloride, tetrachlorethylene, and copper sulfate. These are marketed to the farmer under a suggestive remedial proprietary name. In one instance a commercial house distributed with disastrous results cap-

sules containing a combination of copper sulfate and tetrachlorethylene.

Veterinarians themselves are not entirely exempt from the fault of poisoning due to carelessness or ignorance. In the list of "dangerous remedies" may be included arecaline, eserine, barium chloride, strychnine, copper sulfate, aloin, digitalis, lysol and creolin in uterine douches, and oil in choke.

The symptoms of poisoning vary according to the dosage, the degree of fullness of the stomach, the rate of consumption, the form of the toxic agent, and the species and age of the animal.

FOOD POISONING

While poisoning of animals from the ingestion of food or water thought to contain some unknown infectious or toxic material is often suspected, definite proof of the presence or nature of such material is infrequent. In man most food poisonings are due to bacteria or their toxins. Foremost among these are the paratyphoid-enteritidis group, and the toxins produced by *Clostridium botulinum*. In animals, botulism is the only food poisoning of which there is definite knowledge, and the diagnosis of field cases of this disease presents many difficulties. Yet there are instances, especially among horses, where deaths are apparently due to the ingestion of spoiled food or water, and a thorough investigation fails to reveal the essential cause. There is need of extensive research on the subject of suspected food poisoning in animals.

The term "forage poisoning" is often applied to cases of paralysis in animals when it is suspected that the food or water contains the causative agent.

In 1900 Pearson[1] reported a few cases of poisoning in horses caused by eating mouldy silage. Two horses fed experimentally contracted the disease. The symptoms were paralysis of the pharynx and general paralysis, but *"no brain symptoms."* Until this time it had been customary to apply the term cerebrospinal meningitis to all cases showing similar paralytic symptoms. Since there were no brain symptoms and no lesions of the brain or cord were found on postmortem, Pearson objected to the name cerebrospinal meningitis, and substituted therefor a newly coined term, *forage poisoning*, based upon the demonstrated, but unknown, poisonous substance in the food. This name was selected because of the similarity of the cases to sausage poisoning (botulism) in man. Since the introduction of this term it has been applied to cases presenting similar paralysis, without any knowledge that the food was actually poisonous, and without any postmortem examination; it has been extensively applied to cases presenting any nervous symptoms

whatever, especially brain symptoms, a reversal of its use by the author. At the present time the word is often used for purposes of identification, without regard for either the symptoms or the cause. As used by Pearson, the term forage poisoning means a *paralysis caused by the ingestion of poisonous substances formed in the food.* There are "no brain symptoms," no central motor irritation, and no lesions of the brain or cord on postmortem examination. In the use of the term "no brain symptoms" Pearson apparently meant no disturbance of consciousness. Paralysis of the throat is a common symptom of any form of encephalitis. In this connection the statement by Osler that "there may be 'death in the pot' from many causes" is especially appropriate. So far as animals are concerned, only one of these causes has been identified, and that is the botulinus toxin, and the symptoms of botulism are characteristic. One may surmise that toxic substances may enter the food or water through decomposition of food, bacterial activity, seepage from decomposing organic matter, as a basement well, water that has been stagnant for weeks in a filthy trough, or water that stagnates in swampy holes and pools. While there is no positive proof of the specific etiological agent in such instances, there is an abundance of proof that consumption of food and water from such sources may be fatal. Extensive research is needed to determine the frequency, symptomatology, and pathology of food poisoning in animals. It is probable that many sporadic and slightly enzootic cases of so-called forage poisoning are actually encephalitis; this is especially true of cases showing deranged consciousness and paralysis. In extensive outbreaks of disease characterized by deranged consciousness, any form of forage poisoning is improbable. Until the facts are known, it is less confusing to diagnose according to the symptoms or lesions that are obvious than according to some cause that may merely be suspected to exist in the food or water.

Shortly after Pearson's observations, McCarthy and Ravenel[2] reported a similar disease in horses, and veterinary literature contains numerous similar descriptions. But the vast majority of such outbreaks are never reported. As currently used, the term "forage poisoning" in animals corresponds closely in meaning to the term "food poisoning" in man, but the essential causes of forage poisoning, using the term in a broad sense, are virtually unknown.

Disease characterized by paralysis is often observed by veterinarians. In endemic paralysis, stagnant water from a filthy trough, a basement well, or swampy pastures is often suspected to contain the cause of the undiagnosed disease. In other cases, mouldy or spoiled food is under suspicion. Sometimes the food or water is suspected when the

symptoms clearly indicate an infection of the central nervous system, or a metallic poisoning; for this error there is usually little excuse. In every case, even when the food or water is obviously contaminated, one should be conservative in making a diagnosis of "forage poisoning."

One needs to consider that nonvirus encephalitis (p. 293) is widely prevalent, especially in equines, and that little is known of the cause, or whether it is due to a variety of causes.

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BOTULISM

Definition.—Botulism is a rapidly fatal motor paralysis resulting from the ingestion of toxins produced by the activity of *Clostridium botulinum (B. botulinus)* in decomposed animal or vegetable matter. It is the only food intoxication in animals of which there is definite knowledge. The pathological changes are unknown.

Horses are chiefly susceptible. In parts of South Africa there have been enzootics in cattle (*Lamsiekte*)[1] due to the eating of bones, and a similar disease in cattle (*Loin Disease*) has been described in Texas by Schmidt.[2] Botulism in sheep and cattle in Western Austrialia has been described by Bennetts and Hall,[3] who state that "for many years it has been responsible for a considerable animal mortality in cattle and horses. It was first observed in sheep in 1928, and during succeeding years was considered by officers of the Stock Branch to be a source of greater economic loss than all other sheep diseases." Guinea pigs are highly susceptible. Because of the difficulty in making an accurate diagnosis in sporadic and scattered cases of fatal paralysis in horses, little is known of the general prevalence of disease in horses due to the botulinus toxin. In an extensive report by Meyer and associates[4] the Commission writes concerning its prevalence in animals that "in applying these critical considerations to the accounts of forage poisoning in horses and mules either published or collected by the Commission, it must be concluded that the toxin of *B. botulinus* was probably the cause of equine botulism in the following four instances": 5 burros in Colorado after eating garbage containing canned string beans or spinach; 40 mules being fed on silage in Kentucky; 9 horses and mules fed on corn ensilage in Illinois; and 2 horses after eating a jar of home-canned corn in California. While botulism is often suspected, the infrequency of reports of detection of botulinus toxin in

suspected foods suggests that the disease is infrequent in animals.

Etiology.—*Cl. botulinum* is a spore-bearing anaerobe which was discovered by van Ermengen in 1897. Two distinct types have been recognized in this country. Type A is widely prevalent in California, while Type B is the chief form in the middle and eastern parts of the United States. It is abundantly present in cultivated and virgin soils, and in Meyer's[5] experiments it was shown to multiply in sterilized and non-sterilized samples. Spores were found in the feces of healthy horses, cattle, and garbage-fed swine. He[4] writes that "the bacterium is ubiquitous in the soil and vegetation of almost every State in the Union." Yet "during the past year (1921) we have repeatedly made an attempt to extract from the suspected food the poison of *Bacillus botulinus*, but we have never succeeded. . . . We have been utterly unable to demonstrate the presence of poison in suspected food." Experimentally Meyer inoculated damp hay with 500 million spores of *B. botulinus*, Type A, and incubated it for 27 days. At the end of this time the brown partially decayed hay was found to be exceedingly toxic to guinea pigs; it killed in from 24 to 36 hours to 5 days. These observations proved that the tightly packed and damp hay when inoculated with *B. botulinus* becomes toxic even in the presence of air and in the presence of other aerobic or anaerobic bacteria. The toxic hay was placed in a tin container and exposed to sunlight and rain for 20 days, and when fed to guinea pigs, still proved to be toxic. Meyer concluded that "in the light of these experiments the existence of *B. botulinus* toxin in hay stacks is quite possible."

In Western Australia Type C is regarded as the only type which invades carrion. Rabbit carcasses are usually invaded from the soil, less often from spores carried in the intestines previous to death, and they are highly toxic, even at the end of six months. Contaminated water becomes innocuous to sheep at the end of twenty-five days and water may be detoxified by the addition of 0.2 per cent quicklime. Laboratory tests of suspected water have given inconclusive results.

In 1917 Graham and associates[6] experimented with oat hay which had caused a sporadic outbreak of forage poisoning. The hay was placed in a barrel, water was added, and horses were allowed to drink the water. As a result four horses developed paralysis and died. From the water, anaerobic organisms were cultured, and a sterile filtrate of a broth culture of this organism proved fatal to horses after ingestion. Botulism antitoxic serum administered intravenously protected horses against a lethal quantity of the filtrate administered by the mouth. A similar experience with silage has also been described by Graham.

Theiler and Robinson[7] have described botulism in mules in South Africa, where outbreaks of the disease had prevailed over a period of at

least thirteen years. The toxin was found in chopped fodder containing dead rats which had passed through the machine without being recognized. Injection of two horses and two mules with blood from a dead mule caused death in all four animals. The authors explain this result by the fact that animals ingest not only the toxin, but the bacilli which produce the toxin, and in dead animals these bacilli multiply rapidly and produce more toxin. *B. botulinus* does not enter the blood stream during life. Toxins formed in meat broth cultures were fatal to guinea pigs in doses of 0.0001 cc. of a Berkefeld filtrate, when given subcutaneously. To produce symptoms by means of oral administration, 0.1 to 1 cc. of the filtrate was required. To produce symptoms in a horse, either 20 cc. of the beef-broth culture, or 200 cc. of the filtrate was required, when administered orally. The virulence of the toxin was not reduced by direct exposure to sunlight for 36 hours, or by heating at 60°C. for one hour; it was rendered inactive by exposure to heat at 80°C. for one-half hour.

Morbid Anatomy.—There are no known pathological changes. The chief diagnostic characteristics are the symptoms and the negative postmortem. Decubital injuries are often present on various parts of the body. There may be a few hemorrhages in the trachea and various degrees of hyperemia and edema of the lungs. Ecchymoses are sometimes found on the pericardium, and the meninges of the brain are usually congested. Neither macroscopic nor microscopic changes have been found in the central nervous system. When the patient has survived for several days there may be a secondary pneumonia.

Symptoms.—After experimental administration of botulinus toxin, the horse or guinea pig may develop paralysis and die within 24 hours. The onset of the symptoms is within a few hours to four or five days after ingestion of the toxin, and in exceptional cases the interval may be as long as a week to ten days.

Usually several animals are attacked at about the same time. The dominant symptoms are muscular weakness and paralysis, and as a rule the latter is rapidly progressive, terminating in death in from a few hours to three or four days. Paralysis of the pharynx and tongue is not infrequent; the horse is unable to swallow and the tongue hangs from the mouth. In a few of the described attacks there has been a general weakness over a few days, leading to complete paralysis. In the outbreak among forty mules in Kentucky, the paralytic symptoms were muscular incoördination, weakness, paresis of the pharynx, and recumbency. One mule died in approximately twenty hours, while the others gradually recovered; unsteady gait was observed in three animals for approximately six weeks.

Theiler described the disease in mules as either acute or peracute, less

frequently subacute or chronic. In the latter forms the patient might be able to arise with assistance and move about somewhat with difficulty but soon it becomes recumbent. The consciousness remains entirely normal, and the reflexes originating in the brain and spinal cord retain their function. Even after the animal is down, the ears and eyes respond to the approach of people and other animals. The sensation of the skin is retained, and with the exception of constipation, the bowel and bladder evacuations occur in a normal manner. Theiler accepts the theory that the paralyzing effect of the toxin is upon the end-plates of the motor nerves, the action being similar to that of curare. As in poisoning with curare, the respirations in botulism are increased from the first and they become increasingly more difficult until death results from asphyxia.

The symptoms observed in sheep in Australia are described as follows: "In all cases there is interference with locomotion, but it is doubtful if actual paralysis of locomotor muscles ever occurs. In acute cases sheep can rise and walk, or even run up to a few hours before death. In the advanced stages of more chronic cases, sheep are unable to rise, but this appears to be due to general weakness. Some excitability is noticed in early stages, when sheep will often run blindly into fences, etc. It would appear as if the brain were affected. Even recovering animals will walk away from the flock and cannot be driven with it. The head may be held to one side, the gait is very stiff, and after a short walk may show definite incoördination. . . . Lateral tail switching is very common. Salivation and serous nasal discharge are often seen."

The general examination reveals normal mucous membranes, normal pulse until towards the end, and a normal temperature. There may be sweating. The course varies in different outbreaks; according to Records and associates,[8] it is often prolonged to seven or fourteen days. The mortality is reported at from 70 to 100 per cent. In the differential diagnosis it is of first importance to distinguish botulism from affections characterized by deranged consciousness, motor irritation, and deranged sensation, symptoms which indicate disease of the central nervous system. To make a positive diagnosis of botulism it is necessary to establish the presence of the toxin in the suspected food. The simplest field method of establishing this fact is to obtain an animal from another farm and feed it upon the suspected diet. As a rule when horses die after showing symptoms of botulism, no evidence of the presence of the toxin can be found.

Control.—No effective remedy has been found. Antitoxin is an effective prophylactic. Vaccination with botulinum toxoid confers a high degree of immunity that lasts for at least one year.

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ARSENICAL POISONING

Acute Poisoning.—This is the usual form in domestic animals. It is due, as a rule, to Paris green (acetoarsenite of copper), or white arsenic (arsenic trioxide), or sodium arsenite, or sodium arsenate. In the past, arsenic has been among the more frequent causes of accidental and criminal poisonings because of its use as an insecticide, as a parasiticide, as a poison for rodents, as a weed-killer, and as a stable remedy. Formerly it was a common practice among stablemen to dose horses with Fowler's solution or white arsenic to "improve the condition," and to "cure heaves." Before the days of improved construction of smelter stacks, fields were sometimes contaminated from arsenic-containing fumes, the deposit of arsenic on the vegetation causing chronic poisoning. In recent years arsenic has been displaced by other agents and consequently is a less frequent cause of sickness and death in animals. Paris green is still a relatively frequent cause of poisoning. It may be consumed accidentally where a potato field joins a pasture; on reaching through the fence to graze upon marginal grass an animal may obtain a fatal amount if the vines have been recently sprayed. Cattle may gain access to boxes of Paris green carelessly left in the field, to buckets or barrels in which it has been mixed, or to a dump in the pasture where rubbish has been thrown. When deliberately given by criminals, it may be placed in the grain bin, the mangers or on the fields.

Solutions containing 1 per cent white arsenic have been widely used in Europe to kill lice and other insects on the skin, and numerous cases of poisoning have resulted from the use of more than 500 cc. of the

solution at one time, and from application to an abraded skin. Such preparations are more dangerous when the solution or the animal or the temperature is warm, and when application is followed by rubbing. White arsenic has also been mistaken for salt. The death of 200 cattle after dipping in a solution containing double the usual strength of arsenic and then transporting by train has been mentioned by Kinsley.[1] Pollack[5] has also reported on poisoning in cattle in relation to dipping. He writes that while resorption through the skin may occur, he believes the majority of cases are due to drinking the arsenite of soda solution. White[8] has reported 12 cases with 10 deaths in cows after gaining access to Canadian thistles that had been sprayed with a 10 per cent solution of sodium arsenate. Lamont[3] observed several deaths in horses and cattle that had drunk water from a tank which had been used previously for mixing an arsenical dip. In our own experience arsenical poisoning usually has been caused by Paris green. Other possible sources of arsenic are paints (mineral green, arsenite of copper, emerald green) and aniline dyes. Cases have been reported in which foals have been fatally poisoned from suckling poisoned dams. Drugs such as antimony and tartar emetic also contain arsenic. Arsenate of lead is one of the most toxic and frequent forms of mineral poisoning, but the dominant symptoms and lesions result from the presence of lead. In the cases of arsenical poisoning described by Edds,[10] alfalfa was contaminated from the dusting of an adjoining cotton field, a cow grazed next to a field that had been sprayed, a horse grazed on a field that had been dusted with lead arsenate, a cow and a calf grazed in a field where toxaphene and arsenic had been used to poison grasshoppers.

The fatal dose of arsenic varies widely according to its purity, the degree of fullness of the stomach, and whether it is in solution or in the form of a fine powder. In solutions and in fine powder it soon induces general poisoning. Ruminants are sometimes more resistant than other animals because of the mass of material in the rumen. Arsenic may contain impurities, the most frequent of which is gypsum. Because of these variations, there have been variable opinions concerning the minimum fatal dose. According to Fröhner,[4] a horse may succumb to 3 grams or survive as many as 30 grams. He lists the fatal dose of white arsenic in grams as follows:

	Oral administration	Absorption from wounds
Cow	15-30 grams	2.0 grams
Horse	10-15	2.0
Sheep and goat	10-15	0.2
Swine	0.5-1.0	0.2
Dog	0.1-0.2	0.02
Fowl	0.05-0.1	0.005

Pollack[5] states that a quart of arsenical solution containing 32 grains of sodium arsenite will cause death in cattle in 48 hours in the majority of cases. Clough[6] gives the fatal dose of arsenious oxide administered orally as from 150 to 700 grains (10 to 47 Gm.) for the horse, and from 225 to 700 grains for the ox. He reports that Green and Dijkman gave a horse 15 grains and another 30 grains (2 Gm.) daily for two weeks with no ill-effect. Another horse received 60 grains daily and died after receiving 300 grains. In solution, as sodium arsenite, 15 grains may kill, 35 grains is likely to kill, and 45 grains is certain to kill.

Morbid Anatomy.—The chief lesions are those of severe hemorrhagic gastroenteritis. When the attack has been suddenly fatal, there may be no other visible gross lesions. The gastric mucosa is congested, swollen, and hemorrhagic, and there may be areas of erosion. After eating Paris green, this substance may be found in the stomach contents. In cattle the caustic action may perforate the abomasum or the rumen. As a rule the gross appearance of the spleen, liver, and kidneys is normal. Pollack noted a variable condition of the liver; it might be lighter than normal, yellowish, distinctly yellow, or yellowish-brown. Microscopic examination reveals fatty degeneration in the intestinal and gastric glands, the spleen, liver, and kidneys.

In a herd where several cows had died of arsenical poisoning in the Summer of 1943, Dr. Fincher observed unusual preservation of the tissues of the kidneys and liver when exposed to summer heat that would have caused advanced decomposition in normal organs; it is possible that this may be a characteristic of great diagnostic value. Submucous edema may be found in the walls of the stomach and small intestines.

Symptoms.—There is usually a history that one or two animals have died recently after a brief illness, or have been found dead. Others may be extremely sick. The onset is sudden with marked prostration, staggering, trembling of the muscles and muscular twitching. Marked distress is shown by rapid breathing, restlessness, colic, and groaning. In ruminants there may be salivation and vomiting. Death may occur within three or four hours. When less of the poison has been taken the course may continue for two or three days to a week in the form of a severe, painful gastroenteritis, usually with purging. On examination of such patients one finds complete anorexia, prostration, grinding of the teeth in cattle, mucous membranes congested, dilated pupils, pulse above 100, respirations about 30, and a normal temperature or a fever of 103° to 104°F. The bowel evacuations may be fetid and bloody, the peristalsis is suppressed, and in some cases there may be increased thirst.

Ramsay and Seddon[7] poisoned cattle by feeding the dried and ground leaves of maize eight months after it had been sprayed with a solution of arsenic. For purposes of chemical analysis, they observed "that in animals which are showing symptoms of arsenical poisoning (e.g., diarrhea), the feces, urine, and large bowel contents are specimens which might be expected to show relatively high figures on analysis." Of the internal organs of animals that have been poisoned, the stomach, large bowel, liver, and kidneys contain relatively large amounts of arsenic.

Treatment.—BAL (British anti-lewisite, Dimercaprol) developed in World War II as an antidote to the arsenical blister gases, appears to be the most promising treatment for systemic arsenical poisoning. It is prepared in a 10 per cent solution and the dose is 1 cc. of the solution for each 50 pounds weight intramuscularly every four hours for the first two days, four injections on the third day, and injections twice daily thereafter for ten days or more. Of ten cases reported by Edds,[10] six recovered.

Medicinal treatment consists in the administration of protectives, such as large doses of bismuth subnitrate, or tannic acid. To combat circulatory weakness and collapse, give caffeine sodiobenzoate 2 to 4 drams (8-16 Gm.) or camphorated oil, or black coffee. Atropine 1/4 grain (0.0162 Gm.) is recommended to control intestinal cramps and pain.

Chronic Arsenical Poisoning is rare in animals. Reports by Harkins and Swain[9] on the "*Anaconda Case*," contain extensive information on contamination of vegetation by smelter smoke. The following outline of the more prominent symptoms in horses and cattle, as described by Dr. D. E. Salmon, is quoted from one of these reports:

"*Horses.*—Raised red line at the base of incisor teeth; breath of a garlic odor; loss of spirit, vigor, and endurance; falling of hair; retention of old hair; ulcers of the nose; weakness and imperceptibility of pulse; erosions of the outer side of gums; puffiness above the eye; rough lusterless hair; partial paralysis of hind limbs; with more acute form; (*a*) difficult breathing, (*b*) labored action of heart, (*c*) dilation of the pupils of the eyes, (*d*) partial paralysis of the diaphragm and costal breathing.

"*Cattle.*—Shrinkage of milk within a day or two after smoke has been over pastures; salivation and drooling; constipation; rough scurfy coat; eyes red, inflamed, and weeping; loss of appetite; diarrhea when disease becomes more pronounced; tucked up abdomen; loss of flesh; weakness, loss of vigor; cough; breath of garlic odor; droppings covered with mucus; abortion and failure to breed."

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LEAD POISONING

Etiology.—Lead poisoning is by far the most common form of metallic poisoning in animals. This is explained by the wide distribution of lead, the variety of lead preparations used in agriculture, the extreme susceptibility of animals to its action, and its ability to form organic compounds with plants. Lead combinations of chief importance in toxicology are lead oxide, red oxide of lead, white lead, lead acetate (sugar of lead), and arsenate of lead. Since the action of lead is cumulative, toxic action results when the total amount of small daily doses approximates that of the single toxic dose.

In direct contact with the mucous membranes, lead acts as a corrosive. After resorption it acts as an irritant to the nerve centers, especially upon the psychic and motor centers of the cortex of the cerebral hemispheres, and upon the vasomotor centers. It causes a degenerative atrophy of the peripheral motor nerve endings which results in paralysis of the striated muscles. In chronic lead poisoning in the horse, this effect upon the recurrent laryngeal nerve is marked. The soluble lead salts are resorbed rapidly and excreted slowly. Among the domestic animals, cattle are especially sensitive. In calves fatal poisoning may result from ingestion of very small amounts.

Arsenate of lead is one of the chief causes of lead poisoning. While this compound contains arsenic its toxic action is due to the lead. Animals often gain access to barrels and tubs that have been used for mixing spray, and such containers may be used for feeding stock. I have personal knowledge of two instances of poisoning from the use of such buckets for feeding calves. Such containers may also be deposited on a dump in the pasture or left within reach of animals in the barn. Grass in orchards that have been sprayed may be fatal within twenty-four hours to animals that graze upon it while the spray is still wet. Grazing in orchards during the fall and winter may cause chronic lead poisoning. And hay harvested from orchards or fields that have been sprayed may cause chronic poisoning which develops within six to eight months after beginning to feed the contaminated hay—MacKintosh.[1] In an experiment to determine the danger to cattle from arsenate of lead spray, Paige[2] observed that the feeding of 1 gram of arsenate of lead daily to a cow caused symptoms of poisoning after 26 days, and death in 40 days, when 29 grams had been consumed. A second cow (weight 540 pounds) developed symptoms after 23 days when fed 0.5 gram daily, and purging was violent after receiving a total of 16 grams. A third cow received 28.35 grams at a single dose in a gelatin capsule and suffered from severe, acute nonfatal poisoning. A fourth animal received 56.7 grams in a capsule and died after about 70 hours. Paige[2] estimated that with three sprayings a season, 0.9 of a pound of dry arsenate of lead would eventually reach the ground beneath each tree. Seddon and Ramsay[6] caused death in a sheep with as little as 60 grains of lead arsenate.

Veterinarians are sometimes asked concerning the danger from grass or hay in a field adjoining an orchard or other fields where spraying has been conducted. It is obvious that the wind may carry a dangerous amount of spray to adjoining fields. But a chemical analysis of suspected forage or material would be required to determine whether it contained a dangerous amount of lead. Because of the general use of arsenate of lead spray on vegetables, such as cauliflower, celery, etc., the feeding of refuse from gardens is a possible source of lead poisoning. Other infrequent sources of contamination of forage are streams, which may carry lead from smelters or mines, and smoke from smelters. Animals may be poisoned from drinking water or from grazing on fields that have been overflowed or that have been contaminated by smoke. Reports on this condition have been made by Haring and Meyer,[3] and by Holmes and associates.[4] Cotton[5] has reported the loss of several experimental animals that were poisoned on grass cut from around buildings painted with a sprayer.

Paint is the most frequent cause of lead poisoning in our ambulatory clinic. On one farm a pan containing white lead had been thrown in a small yard where it was covered with a large pile of wood. The yard was used as a night pasture for cows, and when the wood was removed the cows consumed enough of the dried white lead to suffer severely from acute poisoning. In another case a narrow board from an old painted picket fence was used in the partition of a calf pen; from this board, not more than three inches wide and almost black with age, the calf was fatally poisoned. A calf was placed in a box stall where mature cattle had been kept for years; in about a month it licked enough old paint from the walls to cause death. Calves that have access to painted window sash will obtain fatal amounts of lead, even when it is barely within their reach. Epidemics of lead poisoning follow the use of lead paint on box stall partitions. In one case several deaths in purebred cows resulted from throwing a discarded paint pail into a pasture after the painting of a gas station. Cattle have been poisoned from licking paint from advertising boards that were erected in pastures. On one farm several cows died of acute poisoning after the watering trough had been used to wash out old battery boxes; the mud under the outlet of the trough contained pure lead oxide. Lead acetate has been mistaken for salts.

Morbid Anatomy.—In *acute poisoning* the most striking and constant change in cattle is in the abomasum and small intestines in the form of an acute hemorrhagic gastroenteritis. Often the liver presents changes that are almost pathognomonic, in the form of an intense yellow color and a marked degeneration. The parenchymatous degeneration may be so marked that the cut surface presents an appearance similar to that of a hand bath sponge. When the course has been rapidly acute there may be no apparent gross changes in the liver. Under the microscope the liver and kidneys show a marked parenchymatous degeneration. The most intense and constant gross changes are in the stomach and duodenum. In one case in a sheep, described by Seddon,[6] there was an agonal perforation of the abomasum. In horses that have died as a result of chronic lead poisoning, there is found a subacute or chronic mechanical pneumonia with the formation of pulmonary abscesses and gangrene; gangrenous ("vagus") pneumonia is the immediate cause of death. The laryngeal muscles are atrophied.

Symptoms.—The general symptoms vary somewhat. In all acute cases the onset is sudden and the course relatively short. In severe poisoning, prostration, staggering, or inability to rise are prominent symptoms. The mucous membranes may be either normal or congested. As a rule the temperature is normal, but it may be as high as 104°F.

within forty-eight hours after the poisoning. In one group the temperatures were from 103° to 105°F. The pulse is always fast and weak. The respirations may be normal or rapid and shallow. The extremities are cool.

In acute lead poisoning there are two distinctive groups of symptoms: gastroenteritis due to the caustic action of the chemical upon the mucosa, and cerebral symptoms arising from the effect of the lead upon the nervous system. Cattle often manifest excitation of consciousness and motor irritation, and these are of great assistance in making a diagnosis. Poisoned cattle walk in circles, and run into objects because of blindness, and often bellow as if terrified; there is a change of voice. This syndrome we have observed in cattle chiefly in cases of lead poisoning, and while it is possible that it may occur in other diseases, in our experience its presence justifies a provisional diagnosis of lead poisoning until some other cause can definitely be found. The animal may stand with the head pressed against the wall or manger. Epileptiform attacks or muscular twitchings are occasional. Cerebral symptoms have led to a diagnosis of meningitis, and this diagnosis may be erroneously confirmed on autopsy by the presence of congestion and petechial hemorrhages on the meninges. Other nervous symptoms are grinding the teeth, rapid chewing of the cud, and a "kink" in the neck from contraction of the lateral cervical muscles. In one case there was a lateral curvature of the spine as well as the neck. Others fall suddenly, stiffen the legs and suffer from convulsions; these attacks may be repeated every few minutes. There may be champing of the jaws and snapping of the eyelids. As a rule the pupils are dilated. The nervous symptoms of tetany in calves may closely resemble those of acute lead poisoning.

The digestive symptoms are complete loss of appetite and paralysis of the digestive tract. Often evidence of severe gastro-intestinal irritation is present in the form of recumbency and prostration, painful expression, groaning at each expiration, grinding of the teeth, and salivation. The feces are thin, watery, and sometimes fetid; tenesmus has been observed. Some writers mention suppression of the bowel evacuations as one of the symptoms of lead poisoning. I have observed this condition only once and this was in cows severely, but not fatally, sick from licking dried white lead.

Acute lead poisoning in horses is less frequent than in bovines, and the symptoms are somewhat different. In a group of horses that had been criminally poisoned by placing arsenate of lead in the grain, the first symptom was complete anorexia. Examination at the end of forty-eight hours revealed marked depression and a lowered head, as if half

asleep; the bowel evacuations had been normal. The peristalsis was increased and rumbling, pulse 60, mucous membranes normal. There were no signs of pain or distress. Within twenty-four hours the horse was dead. A second horse died in the same manner. Autopsy revealed gastritis and large hemorrhagic patches on the intestines. These horses were treated for "mild indigestion," and the deaths were not expected. MacKintosh[1] refers to three horses that occupied a field which was sprayed late in the afternoon. The following morning one horse was down and unable to rise, while the other two were affected with partial paralysis in the form of knuckling. In general the symptoms are those of an acute painful gastritis, with paralysis as shown by knuckling of the joints. Convulsions may occur, as in cattle.

Chronic lead poisoning (saturnism) in horses has been observed many times, chiefly in districts where there are smelters or lead mines—Macindoe.[7] Lead may be taken in with the food or water, or it may be inhaled in the dust. Paralysis of the laryngeal muscles and inspiratory dyspnea ("roaring") is the syndrome upon which chief emphasis is placed. Laryngeal paralysis is the most constant symptom of chronic lead poisoning in the horse, but often there are other symptoms, and the lead may not originate in smelters. In the cases described by MacKintosh, for example, it resulted from eating hay that had been harvested from sprayed orchards, and from grazing on such orchards in the fall and winter. The hay was fed over a period of six to eight months before symptoms developed. The condition might be first recognized by choke arising from paralysis of the throat, or by a convulsion. When such horses are moved, inspiratory dyspnea becomes extreme, and in contrast to the usual form of roaring it is not immediately relieved when the horse comes to rest. MacKintosh mentions a paralysis of the lower lip, and this condition is shown in the report by Haring and Meyer (Case 10, p. 488). A case observed by the author, showed extreme distress from inspiratory dyspnea following exercise. Another important symptom, upon which insufficient emphasis has been placed, is mechanical pneumonia. This results from paralysis of the pharynx or larynx ("vagus pneumonia") and is in the form of gangrene or abscess formation in the lungs. These lesions may be located in the large lobes and the course of the pneumonia seems to be somewhat longer than in the usual inhalation type. Affected individuals are poor and weak. MacKintosh reports that this form of poisoning occurs in horses grazed in orchards during fall and winter and causes more deaths than any other type. There develops a pharyngeal paralysis with green discharge from the nostrils and frequent choke. Sensory paralysis is also men-

tioned by MacKintosh, who was able to perform tracheotomy without the use of an anesthetic.

The *mortality* in acute lead poisoning is high, often 100 per cent. I have never observed a recovery in a calf. When only a small amount has been consumed by mature cattle recovery is possible. The *diagnosis* of acute lead poisoning is relatively easy, especially in cattle. It has been mistaken for meningitis, indigestion, and "forage poisoning." One case in a calf that was diagnosed by us as lead poisoning because of blindness and walking in circles, was pronounced cyanide poisoning (probably from calcium cyanide) by the chemist. For purposes of chemical analysis several pounds of liver are sufficient.

After a clinical diagnosis of probable lead poisoning has been made, and a negative report has been received from the chemist, a question may be raised as to the possibility of a calf being killed by lead in quantities so small that its detection escapes the chemist. In a case reported by the author,[8] an apparently positive case of lead poisoning gave negative chemical results, an experience which according to Professor W. L. Williams is quite possible.

Treatment.—In acute lead poisoning the chemical antidote is diluted sulfuric acid of the sulfates, such as magnesium or sodium sulfate. These form an insoluble sulfate of lead. For the excitation, chloral hydrate or other narcotics are indicated. Camphor and strychnine are recommended for the paralytic conditions. The symptomatic treatment of chief importance relates to the gastroenteritis and prostration. Especially indicated are protectives, such as liquid petrolatum, bismuth subnitrate, and tannic acid. Large and frequent doses of aromatic spirits of ammonia are useful. Atropine sulfate (gr. ¼) is claimed to be beneficial in the treatment of acute symptoms. Sodium citrate has been reported by Kety and Letonoff[9] as useful in the treatment of lead poisoning in man where its administration was followed by a rapid disappearance of toxic symptoms, a fall in the blood lead concentration, and increased urinary excretion of lead. The dose for cattle is 4 to 8 ounces (120-240 Gm.) three times a day.

In 1953 Holm et al.[10] reported remarkable recovery in four of seven calves which were poisoned with orally administered lead acetate and were treated with Ca EDTA (calcium disodium salt of ethylenediamine-tetraacetic acid) known commercially as calcium versenate. The treatment begins from the fourth to the seventh day after the beginning of administration of the poison. All of the calves were blind, and other symptoms were convulsions, hyperesthesia, inability to stand, and dullness. Improvement was marked soon after treatment started. The initial

dose was 1 Gm. per 30 lb. body weight in a liter of saline solution subcutaneously repeated daily.

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SALTPETER POISONING

(Saltpeter, potassium nitrate; Chile saltpeter, sodium nitrate)

Etiology.—In the past there have been numerous reports of poisoning from accidental administration of excessive amounts of potassium nitrate, when it was mistaken for salts or sodium chloride. The therapeutic dose, as formerly used, is 10 to 20 grams, and as little as 50 grams may cause severe poisoning in a horse. In recent years there have been many cases of poisoning from accidental access to sodium nitrate. This chemical is kept in sacks on many farms for use as a fertilizer. Animals may break open sacks where it is kept in storage and within a few minutes consume enough to cause death in a few hours. On one farm a full sack was thrown on a pile of rubbish in a pasture, and several cows died before it was found. Another farmer bought a few bags of salt at auction, and unknowingly brought home a bag of nitrate of soda. When the cattle were salted from this sack, they began to die rapidly. Numerous deaths have resulted from washing empty fertilizer bags in the watering trough. Enough nitrate of soda to cause death may

be eaten from the fields where it has been spread on the grass for fertilizer. One of our clients lost a number of sheep in this manner, merely from their eating that which remained on the grassy margins of a plowed field. They were so ravenous for the nitrate of soda that they disregarded salt and ate into the ground for the last particles. According to Fröhner the fatal amount for horses and cattle is from 100 to 250 grams; for sheep and swine, 30 grams.

In Colorado and Wyoming[1] there have been numerous reports on livestock poisoning by oat hay and other plants containing nitrate. In calves, a dose of 25 grams of potassium nitrate per 100 pounds body weight caused sufficient methemoglobinemia to cause death; this was probably produced by nitrite which is formed from the nitrate in the gastrointestinal tract. It was necessary for a 500-pound animal to eat only about 5½ pounds of hay containing 5 per cent of nitrate to be fatally poisoned. Calves that received pure KNO_3 in water died of typical symptoms of oat hay poisoning, the conversion of hemoglobin to methemoglobin increasing until it reached 80 per cent of the total blood pigment at the time the animal died. The water extract from 6 pounds of oat hay caused death of a 275-pound calf within three hours. The symptoms were dyspnea, rapid pulse, staggering, and collapse. The diagnosis is most readily made by drawing a small quantity of blood into a syringe and observing the color, which is a dark chocolate brown. Methylene blue in doses of 2 grams per 500 pounds (225 kg.) body weight injected intravenously immediately counteracts the effect of the nitrate by converting the methemoglobin into hemoglobin. This disease has also been described in Canada.[2]

On *postmortem examination* the mucous membrane of the abomasum and small intestine is dark red, purple, or cherry-red in color, and it may be eroded from the caustic action of the salt. The intestinal contents are often bloody or brown in color. Congestion and hemorrhage are sometimes found in the bladder and kidneys. The blood is bright red, or brownish red. In peracute cases characteristic changes may be absent. For examination by a chemist, blood, the stomach and its contents should be submitted. In cattle that died of oat hay poisoning the most characteristic finding was dark chocolate brown blood in which most of the hemoglobin had been converted into methemoglobin.

Symptoms.—Both forms of saltpeter cause severe gastroenteritis. The course is rapidly fatal. Cows turned to pasture in the morning may be found dead at night, and cows that are apparently normal at night may be found dead in the morning. The disease begins suddenly with severe colic, salivation, vomiting, sometimes bloating and polyuria. Depression, weakness, and prostration soon appear. The temperature

remains normal. The poisoning ends fatally in a few hours. Lightning stroke and anthrax have been suspected when animals have been found dead in the field without any apparent cause of death.

Treatment.—Methylene blue in doses of 2 grams per 500 pounds (225 kg.) body weight injected intravenously immediately counteracts the effect of the nitrate by converting the methemoglobin into hemoglobin. To prevent the caustic action administer large amounts of mineral oil, or mucilaginous substances which act as a protective. To combat prostration administer stimulants, such as camphor, caffeine, or atropine.

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MERCURIAL POISONING

Etiology.—Mercurial poisoning is relatively infrequent. The most common cause is found in the faulty use of mercurial preparations for medicinal purposes. Cattle are the usual victims, for they are extremely sensitive to the action of mercury in any form. Quicksilver mixed with lard to form an ointment is sometimes applied to cattle to destroy lice. This seems to be a household remedy in certain parts of Continental Europe, and it is used occasionally here by farmers who have recently arrived. In one herd near Ithaca it was applied along the back, and as a result several cows were taken sick and one died. Stevens[4] has described poisoning in five yearlings; this was caused by placing gray mercurial ointment in small holes in the stanchions as a treatment for lice. In a second herd two cows died from mercurial poisoning caused by the application of calomel to the tops of the heads to destroy lice. Deaths in cattle have resulted from the use of freshly disinfected stables. Calomel is especially toxic to cattle. Fröhner states that application of calomel to the eye of a calf for keratitis may cause mercurialism, and that 8 to 10 grams given internally will cause severe toxic symptoms. Experimental work on dogs indicates that when 4 mg. or more of bichloride per kilo have entered the tissues, death regularly occurs. Suspected mercurial poisoning in feeder steers from feeding corn that had been subjected to a mercury fungicide has been described by Boley *et al.*,[1] and mercurial poisoning, caused by feeding seed grain that had been treated with mercury as a fungicide, has been described by Taylor,[2] and McEntee.[3]

Morbid Anatomy.—The lesions vary somewhat, even in a single group of animals that have been exposed alike. As in all forms of metallic poisoning, there is a hemorrhagic gastroenteritis, sometimes with the formation of erosive ulcers in the stomach. Often the mucosa is edematous. The subperitoneal connective tissue is often edematous and sprinkled with hemorrhages. The liver and kidneys are swollen, and the latter may show hemorrhages under the capsule. The lungs are congested, hemorrhagic, and present areas of bronchopneumonia with abscess formation. The skin and subcutis are anemic and the muscles are pale. The blood is dark red and coagulates slowly. Necrotic lesions are often present on the skin. On autopsy, visible changes may be slight or absent.

Symptoms.—Calomel and gray mercurial ointment produce general mercurialism at once, while corrosive sublimate and iodide of mercury first induce a corrosive action which may be followed by general mercurialism. When corrosive sublimate is taken into the digestive tract it causes a rapidly fatal gastroenteritis; when it is resorbed through the skin or from the uterine mucosa it causes general mercurialism. The *general symptoms* of mercurialism are depression, refusal to eat, and an increase in the pulse, respiration and temperature. The *skin lesions,* which follow external application, are itching, loss of hair, and the formation of thick scabs; these tend to localize around the anus and vulva and on the udder. In Stevens' case there was a painful eruption in the sacral region. *Respiratory symptoms* usually appear in all distinct acute cases. There is a cough, often a fetid breath, and finally distinct pneumonic signs. In the cases of calomel poisoning described by Stevens,[4] one cow developed pulmonary hemorrhage and another had nosebleed. Weakness, paralysis, trembling, and delirium are sometimes present. There is a tendency to hemorrhage in all organs, and especially from the mucous membranes of the nose, lungs, and intestines. The course is brief and fatal after swallowing corrosive sublimate. After external application the course of general mercurialism is from one to two weeks and recovery is frequent.

Harvey[5] has described cases of poisoning from the use of mercurial ointment in which the symptoms did not appear until two or three weeks after its administration to the skin. The cattle then developed paralysis, salivation, muscular atrophy and marked abduction of the limbs.

After eating grain that has been treated with mercurial fungicide, pigs develop weakness, vomiting, anorexia, blindness, and nervous symptoms shown by backing, continuous walking, and convulsions. The

temperature may be normal, and the course about a week ending in paralysis and death.

Treatment.—Immediately after corrosive sublimate has been swallowed, administer white of eggs or large amounts of milk to precipitate the mercury. In the treatment of animals one rarely reaches the patient soon enough to use this treatment effectively. Any mercurial preparation that may be on the skin should be washed off. In poisoning with mercurial ointment, prescribe chemicals which form insoluble compounds with quicksilver, such as sulfate of iron or sulfur. To combat the depression and paralysis, give camphor, coffee, or atropine. For chronic mercurialism iodide of potash has been generally recommended.

In a report by Mintz[6] of the Massachusetts General Hospital, on the treatment of twenty-one cases of bichloride poisoning in man, he writes: "It can clearly be seen that all experimental and clinical data show conclusively that little is to be hoped for in treating a case of bichloride poisoning by administration of drugs. So far no specific antidote has been found that will successfully combat the intoxication. . . . To combat this systemic intoxication sodium thiosulfate (sodium hyposulfite) has long been advocated and employed. That this drug is useless has been definitely proved. Melville and Bruger found that the length of life of dogs receiving a fatal dose of bichloride could not be prolonged by subsequent injections of sodium thiosulfate." Chief emphasis is placed on mechanical removal from the alimentary canal before it reaches the portal circuit. Blaisdell[7] has reported recoveries in ten cases of acute mercurial poisoning in man with no deaths; the patients received large doses of sodium thiosulfate. Cenini[8] has also reported on the beneficial action of sodium thiosulfate in combination with dextrose per vein, and bismuth subnitrate per os.

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CYANIDE POISONING

(Prussic Acid; Hydrocyanic Acid; HCN)

Etiology.—Hydrocyanic acid is a colorless volatile liquid possessing a characteristic odor described as that of peach blossoms. The official preparation of the *United States Pharmacopeia* is a 2 per cent solution of this acid. Cyanide is used commercially in the form of a number of salts, such as calcium, sodium, and potassium. Calcium cyanide ("Cyanogas") is employed as an insecticide and for the destruction of animal pests, especially woodchucks. Many species of wild and cultivated plants are also capable of developing hydrocyanic acid. In this case it is formed from various glucosides. In sorghum, which is one of the more common sources of cyanide poisoning in animals, it is believed that the glucoside is split by the enzyme *emulsion* into hydrocyanic acid, dextrose, and benzaldehyde. The following plants are listed by Couch[1] as capable of developing hydrocyanic acid:

Chokecherry (wild), *Prunus virginiana.*
Blackcherry (wild), *Prunus serotina.*
Sorghum, *Sorghum vulgare.*
Johnson grass, *Sorghum halepense.*
Flax, *Linum usitatasimum.*
Arrow grass, *Triglochin maritima* and *T. palustris.*
Velvet grass, *Holcus lanatus.*
Christmasberry, *Photinia falicifolia.*
Sudan grass, *Holcus sorghum, sudanesis.*

The presence of HCN in sorghum plants was discovered in 1902—Vinall.[2] In general, it is reported that the acid develops only when the normal growth of plants has been retarded or stopped by drought, frost, bruising, trampling, wilting, moving, or other causes; that the mature plants contain a smaller percentage of potential acid than do young plants; that those on poor soil contain less than those grown on good soil; that fertilization on nitrates markedly increases the percentage of the acid; that well-cured grass contains little or no acid, and that its development in the stomach is retarded by such substances as alfalfa hay, glucose, corn, and starchy foods. Marsh,[3] writing on the subject of *Stock Poisoning Plants on the Range,* states that "it has been found that the rather widespread idea that cherry leaves are especially dangerous when wilted is erroneous."

Poisoning of stock may be caused by grazing on plants containing cyanide, or by accidental or criminal use of cyanide compounds, espe-

cially calcium cyanide. Since this preparation has come into general use for killing woodchucks and other pests, and has been added to the list of poisons commonly found on the farm, its use in criminal poisoning of stock has been reported frequently. I have personal knowledge of deaths of about fifteen cattle from this cause within one year.

The cyanide-containing grasses and sorghum are poisonous when growing green in the fields, either as a stunted first growth or a second growth. Losses from grazing on plants are more prevalent in the West than in the eastern part of the United States or the South. In writing on this disease in California, Haring[4] reports that losses in cattle and sheep from Johnson grass poisoning are frequent, but complaints of poisoning from sorghums are rare, and that forage is not likely to prove dangerous when grown with an ample supply of moisture. Poisoning of sheep on the Western Range from eating wild cherry leaves is reported by Marsh[3] as definitely proved, although many experiments have been without results. The sheep are poisoned along drives that are bordered with wild cherry (*Prunus nana*) and where there is very little else for animals to eat. "The leaves of the common chokecherry of the West (*Prunus demissa*) are regarded as highly poisonous from early summer until they begin to turn yellow in the autumn" (Nevada).[5] Peters[6] and associates have written on *Poisoning of Cattle by Common Sorghum and Kaffir Corn* in the Midwest, where the dry climate is favorable to the growth of toxic forms of the plants.

The amount of combined prussic acid sufficient to make an animal sick was found by Avery[6] to be 0.4 gram, which would indicate that the lethal dose in mature animals is approximately 0.5 to 0.6 of a gram. It is estimated that the amount likely to prove fatal may be ingested in 18.9 pounds of Sudan grass or in 7.6 pounds of sorghum.

Within the past few years there have been numerous reports from Europe on cyanide poisoning from eating linseed-cakes and linseed-meal. The process of manufacture of linseed-meal is supposed to create sufficient heat to destroy the cyanide-producing qualities. Barr[7] suspected cyanide poisoning in two herds. Two heifers died suddenly and a third was very sick. At first anthrax was suspected. Analysis of the feed revealed a high percentage of cyanide. Autopsy showed gastro-enteritis and pneumonia with an intense congestion of the lungs. Campbell[8] described a similar outbreak in three horses fed a mash of linseed-meal. The symptoms were sweating, dyspnea, frothing at the mouth, paralysis of the bowels and fast pulse. Death occurred in one after 15 hours and in another after three days. On postmortem examination the lungs were found to be filled with dark red blood, and the horse that lived three days had a severe enteritis. According to Quentin,[9]

cyanide has been demonstrated in the seeds, and it can be detected by a chemical examination of the linseed-cakes. The symptoms are spasms, dyspnea, somnolence, collapse, and often sudden death. Gastro-enteritis is frequently present. Autopsy reports mention severe inflammation of the abomasum and intense congestion of the lungs, often with pneumonia. There is a distinct uniformity in the descriptions of the symptoms and lesions as written by different authors. Quentin[9] mentions soda as an antidote. In *Nebraska Bulletin No. 77* it is reported that one farmer saved every case of cyanide poisoning that received soda and vinegar. Udall administered "Cyanogas" to calves experimentally; this is said to contain not less than 40 per cent and not more than 50 per cent of calcium cyanide. The results were as follows:

Calf 1.—About 4 days old. November 11, gave 0.1 gm. orally in a capsule without results. November 12, gave 0.2 gm. in like manner; this caused drowsiness and anorexia. November 13, gave 0.3 gm. orally in a capsule; this caused collapse, trembling, dyspnea, and twitchings bordering on convulsions. November 18, the calf was still sick from previous dosage, gave 0.4 gm. orally in a capsule without results. November 29, autopsy: found extensive multiple abscess formation throughout both lungs.

Calf 2.—November 18, gave 0.2 gm. orally in capsule; severe symptoms followed immediately. November 19, gave 0.3 gm. in like manner; death occurred within twenty minutes. Age about 4 days. A third calf received 0.5 gm. orally in a capsule and failed to show any evidence of poisoning. When 0.5 gm. is shaken in a pint of milk and given to a 4-day-old calf, with a 2-ounce syringe, the animal will collapse after taking about 2 ounces, and it may recover.

Action of Cyanides.—The cyanides act on the protoplasm and suspend the activity of all forms of living matter. Asphyxia is caused by suspension of the exchange of gases between the tissues and the blood. If the action of the cyanide is removed the activities of the tissues are resumed. Since the tissues do not abstract oxygen from the blood in cyanide poisoning, the blood in the veins maintains the arterial condition and is bright red. In the blood, cyanides are rapidly changed into nontoxic forms, which are excreted in the urine and saliva; this change accounts for the difficulty of finding cyanides in the tissues. Concentrated solutions of cyanide salts are corrosive to mucous membranes. Thus an animal poisoned on cyanide compounds may recover from the immediate effects of cyanide gas and succumb to the corrosive action or other indirect results. In the experimental cases previously mentioned, and in other cases diagnosed by the chemist as cyanide poisoning, either corrosion of the abomasum or pulmonary abscess were sometimes found.

It is evident that the action of cyanide compounds is variable.

Morbid Anatomy.—The descriptions of autopsies on cattle that have died of cyanide poisoning after eating cyanide-containing plants do not mention any abnormal condition of the cadaver. The cherry-red color of the blood after exposure to the air is held to be the most characteristic postmortem change. In all of our cases of cyanide poisoning this has been observed. It is especially striking on black rubber or other dark surface after an exposure of several hours to the air. Animals that have died of cyanide poisoning are said to give off a distinct odor of prussic acid on autopsy; this we have never been able to detect.

The corrosive action of calcium cyanide may cause perforation of the abomasum. In two cows and a calf that died after an illness lasting from twelve to forty-eight hours, the chemist made a diagnosis of cyanide poisoning. In each of these cases I found a perforation of the abomasum and diffuse peritonitis. Distinct burning of the mucosa of the esophageal groove was also found in an experimental calf which received 0.2 gm. calcium cyanide (cyanogas) in a capsule. In each of these cases there was an intense gastroenteritis affecting the abomasum and duodenum.

Apparently calcium cyanide may exert an indirect action upon the lungs. This was first suggested in our clinic by a report from the chemist that evidence of cyanide poisoning had been obtained from the tissues of a cow which was killed after a course of five days because of impending death from pneumonia. Both lungs contained numerous abscesses, but we did not suspect any form of poisoning at the time of the autopsy. About a year later a 3-months-old calf died after showing symptoms of cyanide poisoning for forty-eight hours. On autopsy both lungs showed increased firmness throughout and histological section revealed bronchopneumonia. Our experimental calf No. 1 also developed an extreme case of multiple pulmonary abscess. Thus it is quite evident that cyanide poisoning may cause pulmonary lesions, and that the postmortem changes are variable. In two cases there was a distinct turbidity of the lens. When death occurs within a few minutes after the appearance of the symptoms there are no visible tissue changes, with the exception of the cherry-red color of the blood.

Symptoms.—When cyanide-containing plants are grazed, symptoms may appear within ten to fifteen minutes after the animals reach the field, and deaths may occur within a few minutes. In *Nebraska Bulletin No. 77* there is a report of twenty-one deaths in a herd of thirty-two dairy cows within an hour after eating a stunted growth of Kaffir corn. One observes drowsiness, lachrymation, twitching of the muscles, staggering and inability to stand. Dyspnea is marked. A slight diarrhea at

the onset is often present. It is also said to be identified by the odor.

When a toxic dose of calcium cyanide is given, there is an immediate effect from the action of the cyanide gas. The most striking symptom is extreme dyspnea with open-mouth breathing and frothing at the lips. With few exceptions the animal falls and is unable to rise. Extensive muscular twitchings and convulsions are frequent. After the severe symptoms have persisted a few minutes the animal gives a distressed bleat; this crying out takes place near the termination of most fatal cases, and is said to be characteristic of cyanide poisoning in all species, including man. When down and breathing with difficulty there seems to be a tendency to rest on the sternum with the front limbs abducted. At times the head may be turned to the side with the eyes closed, as in milk fever. But in a few minutes the active signs of distress recur with the appearance of impending suffocation; at such times a poisoned calf may stand with the head and neck extended and open and close the mouth, or make chewing movements, or grate the teeth. Nystagmus has been observed. In one case the animal was thought to be suffering from choke, and bubbling râles could be heard over the lungs. A fluid movement of the bowels at the beginning of the attack has been mentioned, but this has been observed in our cases only once. If the animal survives the immediate action of the gas, the acute distress subsides and there may be complete recovery within a few hours. It is sometimes stated that if the patient survives for an hour recovery is certain, but this does not apply to poisoning caused by ingestion of cyanide compounds. After the initial suffocation the symptoms vary rather widely. In one case the calf was blind and walked in circles as in lead poisoning, and this was the clinical diagnosis; death occurred after about forty-eight hours. Another calf died at the end of forty-eight hours of dyspnea. In cases of perforation of the abomasum, the course has been from twelve to twenty-four hours with symptoms of severe acute peritonitis. The digestive tract is paralyzed and the bowel evacuations cease. When pneumonia develops a fatal termination may occur in from forty-eight hours to several days. Dilatation of the pupil with turbidity of the lens is significant. The mortality is high and the course is brief. Only two cases under our observation have survived for more than about forty-eight hours.

Treatment.—Experimental treatment of cyanide poisoning has been reported by Bunyea,[10] who concluded that the most satisfactory of all treatments tried consisted of a combination of sodium nitrite (10 cc. of a 20 per cent solution) immediately followed by sodium thiosulfate (30 cc. of a 20 per cent solution) intravenously. This amount was effective in cattle against two lethal doses of cyanide. Corresponding results

in sheep were obtained from 10 cc. of a 10 per cent solution of sodium nitrite, immediately followed by 20 cc. of a 10 per cent solution of sodium thiosulfate. Stanton,[11] who has treated many cases of sorghum poisoning in cattle in Nebraska, expresses the opinion that any animal, poisoned on any of the sorghums, will recover if given from 40 cc. to 80 cc. of a 20 per cent solution of sodium thiosulfate intravenously any time before breathing ceases.

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SALT POISONING

(Sodium Chloride)

Recent studies carried out at the New York State Veterinary College have established that salt poisoning is a readily recognizable pathological as well as clinical entity. Both the symptoms and lesions are neurological.*

Etiology.—The observations on swine made by Professor Smith include 7 natural outbreaks, 266 animals and 64 deaths. Either extra

* This description is based largely on observations reported through the courtesy of Associate Professor D. L. T. Smith, Ontario Veterinary College, who conducted experiments at Cornell University on salt poisoning in swine.

salt was being fed in the belief that it "was good for pigs" and increased weight gains, or it was added by mistake to feed or water, or the pigs were being given food of unknown chemical composition such as garbage. In all cases the supply of fresh water was limited, usually the only source of fluid was that mixed with food or slop.

In the course of the studies it was established that:

(a) Salt poisoning can be produced in pigs with concentrations of pure NaCl as low as 2 per cent in the slop if water intake is limited;

(b) A 180-pound pig can consume 250 grams of NaCl per day with impunity if an unlimited supply of fresh water is available;

(c) A lethal dose of NaCl given in 20 per cent aqueous solution via stomach tube to 4-months-old pigs, is any amount over 1 gm. per lb. body weight. Doses of 0.8 to 1 gm. per lb. tend to produce epileptiform seizures;

(d) Sodium propionate in 3.6 per cent concentration in slop can produce clinical and pathological manifestations identical to those caused by NaCl. Water intake must be curtailed as in NaCl poisoning. The feeding of slop containing 2.7 per cent KCl plus CaCl did not produce any of the phenomena which characterize sodium chloride poisoning. These facts suggest that the sodium rather than the chloride ion is responsible for the disease.

The influence of high salt intake in cattle has been described by Pistor et al.[1] in Arizona "where range cattle are given access to a mixture of 30 per cent salt and 70 per cent meal supplement. The salt limits the consumption, and range cattle consume from 2 to 3 lb. of the mixture daily during the short grazing seasons. . . . Each year there are numerous reports of death loss due to 'salt poisoning,' and most of these cases occur on ranges with little water. . . . Ruminants can tolerate relatively large amounts of salt if sufficient water is available."

Morbid Anatomy.—The findings are variable and nonspecific. The gastrointestinal tract may be only mildly reddened. Occasionally there is severe gastroenteritis. In pigs that have shown a number of epileptiform seizures gastric ulcers are frequently present. Histopathological changes are meningo-encephalitis characterized by infiltration of eosinophils, necroses, gliosis and capillary proliferation. The lesions are most evident in the gray matter of the cerebral cortex. Later in the course of the disease round cells may be the predominant inflammatory elements.

Symptoms.—The early clinical signs in swine consist of pruritus, constipation, depression, blindness and unawareness of surroundings. In peracute cases, prostration, coma and death may follow within twenty-four hours. In some less severely affected pigs, blindness asso-

ciated with forced circling, involuntary twitching of the snout and clonic spasms of the neck muscles are observed. The animal may stand for long periods with its head or snout pressed against the wall. The most frequent and certainly the most striking phenomenon is the occurrence of epileptiform convulsions. These episodes are always heralded by twitching of the snout, with tonic and clonic spasms involving progressively the muscles of the neck, trunk, forelegs and finally the hind limbs. The pig may move backwards rapidly and assume a dog-sitting position or even turn over backwards. A convulsion lasts up to one minute, and in typical cases ends with profuse salivation, complete exhaustion and collapse. Food and drink are usually ignored. Several experimental clinical cases have been observed to convulse at regular seven minute intervals. The pig may die in a convulsion (respiratory arrest) or the fits may suddenly cease with permanent clinical recovery after a short period of readjustment.

Treatment.—Wauttie[2] gives data to indicate that the blood level of calcium is low in salt poisoning and cites clinical experiences which are purported to establish calcium therapy as a specific treatment. We were unable to substantiate either of these claims. The contaminated food should be taken away and fresh water in small amounts, at first, should be given. Drenching must be avoided as the semiconscious condition of the animal predisposes to inhalation pneumonia. We have found that the majority of our experimential cases recover if they are kept from injuring themselves by providing adequate space and ample bedding.

Prevention.—The amount of sodium chloride in the diet of pigs should be carefully controlled and should not exceed the recommended allowance. A liberal supply of fresh water other than that given with the feed as slop should be available to pigs at all times: this is particularly important if garbage or other food stuff of unknown composition is being fed.

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COPPER SULFATE POISONING

(Bluestone; Blue Vitriol)

Etiology.—Poisoning from copper is relatively infrequent, and poisoning from copper sulfate is by far the most common form associated with this metal. Animals have been poisoned from eating plants and

grain after sprinkling with a solution of copper sulfate. I have personal knowledge of at least three instances where sheep were poisoned from the use of too strong a solution of copper sulfate when drenched for stomach-worm disease. This is usually given in a 1 per cent solution, and its use in a much greater strength may be disastrous. Copper sulfate acts as a corrosive to the mucous membranes, causing severe and rapidly fatal gastroenteritis. Poisoning of sheep from grazing in an orchard that had been sprayed with Bordeaux mixture has been described by Muth;[3] samples of liver from two ewes gave values of 1,078 and 1,340 ppm on a dry weight basis; a clover sample taken three months after the last spray contained 42 ppm, most of which had been taken from the soil by the clover plants. It has been reported from Australia[4] that subterranean clover pasture with a copper content of 12.8 ppm is toxic to sheep. Normal livers rarely contain more than 500 ppm,[5] and may be as low as 16 ppm.[1] Since the losses may continue for as long as five months after the ingestion of the copper, it may be difficult or impossible to locate the source of the poisoning.[1]

Morbid Anatomy.—As seen in sheep there is an intense inflammation of the abomasum and small intestine. Lander[2] reports the condition found in four colts. In addition to gastrointestinal hemorrhage, the liver was swollen, brownish yellow and friable, the spleen was enlarged and the kidneys were congested. The kidneys may be swollen and the tubules filled with bloody casts.

Symptoms.—Shortly after swallowing a strong solution of copper sulfate (10 per cent) sheep suddenly show abdominal pain, vomiting, and purging. After a time there may be collapse and convulsions. The pulse and temperature are high, and the mucous membranes are congested. There is anorexia and there may be great thirst. Jaundice may develop if life is sufficiently prolonged. In horses, copper sulfate poisoning may cause spasm of the diaphragm and dyspnea with prolonged expiration.

Boughton and Hardy[2] have described a form of chronic copper poisoning in sheep in West Texas. It resulted from long-continued ingestion of commercial salt mixtures which contained relatively small percentages of copper sulfate in addition to sodium chloride and tobacco dust. Death occurred within a day or two after the appearance of symptoms. It was characterized clinically by hematuria, inappetence, icterus and extreme weakness. The pulse and respirations were increased and the temperature was normal. The postmortem changes were yellow liver, dark brown or black kidneys, swollen "blackberry jam" spleen, and generalized icterus.

Treatment.—The antidotes for copper sulfate poisoning are mag-

nesium oxide, sulfur, whites of eggs, milk, and dextrose. The course is from a few hours to one or two days and the mortality is high. Borderline cases may receive protectives in the form of bismuth subnitrate in milk, or liquid petrolatum. Stimulants may be prescribed for weakness.

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STRYCHNINE POISONING

Etiology.—Strychnine poisoning is relatively frequent in animals. It may follow immediately after an excessive dose, or it may appear after several large doses have been given, when it is the result of a cumulative action. Often the prescribed dose is only slightly less than the toxic amount, and for this reason prescription poisoning of animals is relatively frequent. Fröhner states that poisoning may result from the use of old solutions; either through evaporation of the water, or crystallization of the strychnine, the solution which remains at the bottom of the bottle may be much stronger than the original. I once prescribed a soluion containing 0.5 per cent each of arsenic and strychnine, and when the owner returned for more he was given a mixture of 2 parts of salts to 1 part each of gentian and nux vomica. He then gave both preparations to a horse in half-ounce doses thrice daily and within three days distinct symptoms of strychnine poisoning appeared. One of our stock prescriptions contains 0.5 per cent strychnine in solution, and at one time this was increased to 1 per cent; within a few days two horses were poisoned, one fatally. Although cows are said to tolerate relatively large amounts of strychnine, I have observed poisoning from a medicinal dose given subcutaneously. There seems to be a wide variation in the susceptibility of cows to strychnine given subcutaneously; apparently the purebred type is more sensitive than ordinary cattle. The fatal dose when administered subcutaneously is given for the horse at from 3 to 4.5 grains, and for cattle 3 to 6 grains. On a basis of my own experience, I would not prescribe orally more than 3.5 grains daily to a large animal.

Action and Symptoms.—The spinal marrow is the seat of the toxic influence in strychnine poisoning. The reflex irritability is greatly increased so that the slightest stimulation by sound, vision, or touch may cause powerful reflex activity. The seizure is sudden, as in epilepsy, though a preference for the muscles of the extremities may be shown. Almost immediately the entire body is involved, the limbs, neck, back, and tail being held in a stiff position. There is an expression of fright shown by the prominent position of the eyes. The spasms of strychnine poisoning resemble those of tetanus, but they come on more suddenly, last for three or four minutes and are separated by intervals of complete relaxation. In contrast to tetanus the muscles of the jaw are rarely involved. The time required for the appearance of the symptoms after subcutaneous administration is only a few seconds; after oral administration the symptoms may appear in from a half hour to six or eight hours. Within two hours from the commencement the case usually terminates in some manner. *Autopsy* is usually negative. Rigor mortis sets in rapidly and the cadaver may be preserved for days.

Treatment.—The traditional treatment for strychnine poisoning is to administer narcotics, such as chloral or morphine. Recently *sodium amytal* has been reported by Kempf, McCallum, and Zerfas[1] to be a highly efficient antidote. Eleven cases were treated with intravenous injections and all recovered. The dosage varied from 7½ to 27 grains, and the effect was almost immediate. LeDret[2] has reported similar action in a dog from the use of Somnifen.

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INSECTICIDES

The development and general use of synthetic insecticides for destroying parasites on animals, materials, and crops, as well as in stables and other buildings, has led to fatal accidents through misuse or misconception of the compounds. Contributions to such accidents have resulted from labeling containers with meaningless commercial names, failure of the label to indicate clearly the nature of the contents, and free access to all buyers. The group most widely used on animals falls under the classification of *chlorinated hydrocarbons*, which are ar-

ranged and briefly described here in the ascending order of their toxicity.

DDT.—This product may be safely sprayed on animals in concentrations of 8 per cent, and it has been widely used in the control of flies. With the development of DDT-resistant strains of flies, however, its effect has largely been lost. Because it is stored in the animal and secreted in the milk, its use on dairy cattle is prohibited. Similar preparations are TDE and Methoxychlor.[2]

Chlordan.—Estimated fatal dose 1 gm/kg body weight.

Calves	1.0 per cent, toxic to young dairy calves.[4]
Cattle	0.5 per cent, ordinarily used.[4]
Cattle	4.0 per cent, not severely harmful.[4]
Pigs	4.0 per cent, tolerated.[4]

Toxaphene.—Estimated fatal dose 60 mg/kg body weight.

Calves	1.0 per cent, may kill baby calves.[4]
Cattle	0.5 per cent, is the usual concentration.[4]
Cattle	0.4 per cent, not severely harmful.[4]
Swine	0.5 per cent, eradicates mange.[3]
Pigs	4.0 per cent, tolerated.[4]

Concentrations of 1 per cent toxaphene and chlordan injured a smaller proportion of sheep than did dips containing 0.06 per cent of gamma isomer of BHC.[3]

Dieldrin.—A recently developed insecticide,[1] p. 52.

Calves	0.25	per cent, sometimes fatal, 1 to 2 weeks old Jersey.
Cattle	0.5	per cent, 3 applications at 2-week intervals, toxic.
Cattle	2.0	per cent, single application, toxic.
Cattle	1.0	per cent, single application, nontoxic.
Pigs, suck	4.0	per cent, tolerated.
Sheep	4.0	per cent, toxic.
Lambs, suck	2.0	per cent, tolerated.
Lambs, suck	3.0	per cent, toxic.

Lindane.—BHC; Benzene Hexachloride, 99 per cent gamma isomer; Isotex.

Calves	0.05 to 0.1 per cent, toxic in 1- to 2-week Jersey calves.[1]
Calves	0.1 per cent, nontoxic in very young Hereford calves.[1]
Calves	0.02 to 0.06 per cent, nontoxic.[1]
	0.03 per cent is the usual concentration.[4]
Cattle	0.25 per cent, 12 applications at 2-week intervals, nontoxic.
Cattle	0.75 per cent, sprayed twice, eradicated scabies.[2]
Cattle	0.06 per cent, sprayed twice, 10 days apart, nontoxic.
Cattle	0.12 per cent, sprayed once, eradicated scabies.[2]
Pigs	1.0 per cent, tolerated.

A second group includes *organic phosphatic insecticides,* which are

highly toxic, and the most widely used compound is included here:

Parathion.—(TEPP; Thiophos 3422; E605; Niran: Alkron; Tetraethyl Pyrophosphate.) This is a highly toxic compound with cumulative action. Special precautions are necessary to prevent inhalation and skin contamination. It has been the cause of numerous fatalities in animals, either from direct application, or being present in stables where parathion spray was used to kill flies, or from exposure in fields where it was sprayed as an insecticide.

Morbid Anatomy.—An extensive acute pneumonia with edema and emphysema of the lungs was observed in a bull that died after a brief attack from parathion poisoning used in the stable as a spray.

Symptoms.—The onset of symptoms appear within thirty minutes to forty-eight hours after exposure with twitching of muscles, excessive salivation, grating the teeth, rolling of the eyes, blindness, and either depression or excitement, or the two may alternate. In violent attacks there is dyspnea with an increase in the pulse, respiration, and temperature which may reach as high as 114° F. Convulsions are severe, frequently repeated, and soon terminate in death.

Treatment.—First control the convulsions by the use of barbiturates,[4] and wash the animal thoroughly with the use of synthetic detergents to remove the remaining insecticide. Atropine, 1/16 to ¼ grain subcutaneously, if given early and repeated as needed, may prove to be a specific in cattle; in man it has received credit for saving desperate cases.[5]

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BOVINE HYPERKERATOSIS

(X-Disease)

In 1941[1] there appeared in New York in widely separated areas a fatal disease of cows and young stock characterized by emaciation, circumscribed areas of proliferative stomatitis, esophagitis, enteritis, and a thick dry skin with loss of hair. Two distinct types were encountered: a less frequent acute rapidly fatal malady, and the usual chronic type, which may be a continuation of the acute, or proceed as chronic from the onset. This disease resembled advanced cases of

mange, but mange mites were not present, and for identification it was termed X-disease, and was generally believed by research workers to be an infection caused by a virus. Finally it was observed in at least 35 states, chiefly in the South and Midwest with no reported cases in New England or on the Pacific Coast.

Etiology.—In 1949 a research program coordinated by the Federal Bureau of Animal Industry was organized in 18 states to study the cause and symptoms of X-disease. In 1950 Olson and Cook[2] proved that there was a toxic substance in feed pellets fed to 150 calves, of which 130 developed bovine keratosis and 47 died; and it was also observed that hyperkeratosis developed in several calves which received milk from an affected cow.[2] In 1952, "Bell[5] of Virginia was the first to

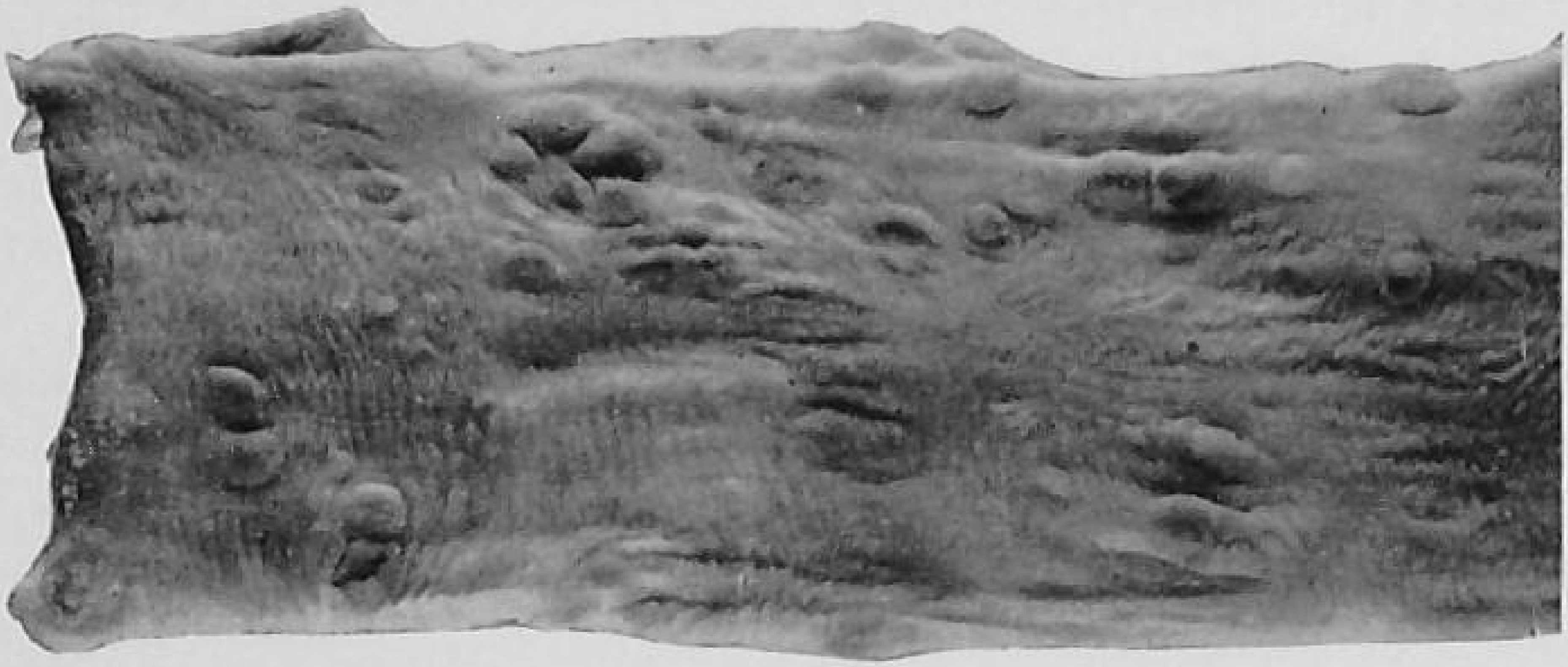

Fig. 108.—Proliferative circumscribed raised lesions on the esophagus; identical lesions appear on the mucosa of the mouth and pharynx. (Courtesy of Peter Olafson)

produce bovine hyperkeratosis with a petroleum product."[2] He observed that calves were licking a lubricant from the springs of trucks, and he produced the disease by oral administration of the lubricant obtained from the farm. He also proved, with the aid of the manufacturer, that the toxic substance was a chemical additive, a highly chlorinated naphthalene compound, which had been used as an extreme pressure agent in the manufacture of the lubricant. These results were also obtained by Sikes and Bridges[4] and by Olafson.[2] In 1953 the U. S. Bureau of Animal Industry[3c] released a statement that "The only proved cause of X-disease is a highly chlorinated naphthalene, used in special purpose lubricants and in certain other products, either as an added ingredient or as a contaminant.

"Individual stock owners have suffered severe losses during the past year from the disease caused by feeding pelleted feed contaminated with grease containing highly chlorinated naphthalene compounds. Feed

manufacturers and oil companies are now cooperating in helping to keep lubricants containing the compounds away from feeds, and potential losses from X-disease or hyperkeratosis in cattle have been greatly reduced.

"Hyperkeratosis is not produced unless the grease contains highly chlorinated naphthalene; it is not produced by solvent extracted cottonseed or soybean meal, by commercial wood preservatives produced in this country, or by insecticides approved and sold in this country for use on cattle or forage. There are many things which will produce local irritation of the skin of cattle when applied frequently over a period

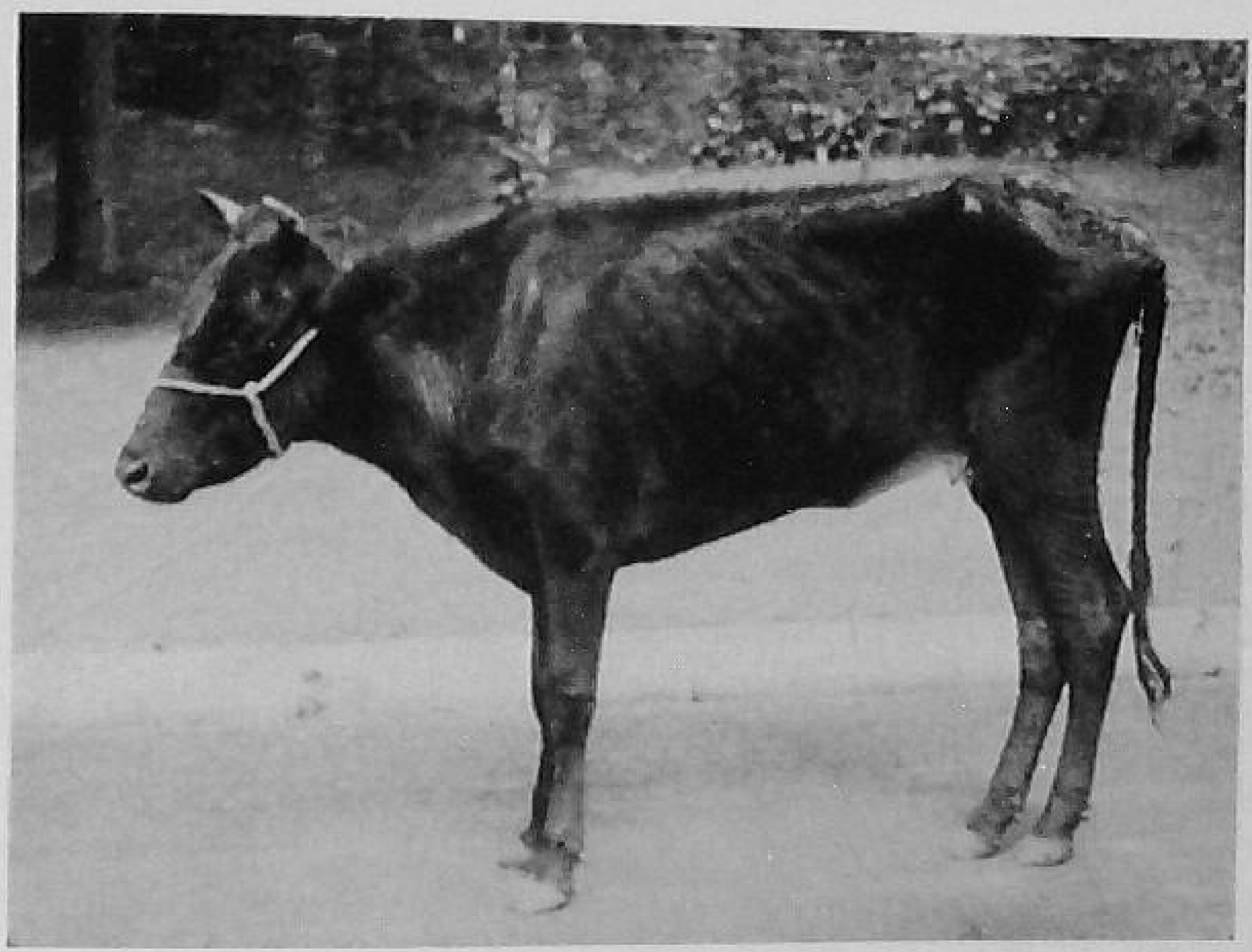

Fig. 109.—Emaciation and thickening of the skin with mange-like appearance. (Courtesy of Peter Olafson.)

of time and which may be mistaken for hyperkeratosis."

Morbid Anatomy.—The changes most commonly mentioned are emaciation, and a dry, wrinkled, mange-like thickening of the skin especially over the neck, shoulders and inguinal region (hyperkeratosis) caused by accumulation of keratinized material on the surface.[7] Proliferative, wart-like circumscribed swellings may be present on the muzzle, the gums, the dental pad, the tongue and in the esophagus, and the salivary glands may undergo squamous metaplasia. Other lesions that have been mentioned are areas of hemorrhage in the walls of the small intestine, ulcers in the abomasum, cystic dilatation of the glands of the stomach, duodenum and rest of the intestinal glands, gastric

edema, and enteritis of the small intestine or cecum.[6] "Cystic dilatation of the tubules in the kidney cortex was the most pronounced change and was observed in all cases."[6] Frequent lesions are squamous metaplasia of epididymides, seminal vesicles, and Gartner's ducts,[3a] mucoid papillary proliferations in large bile ducts and gall bladder, fibroses and duct proliferation in the pancreas, proliferation of small bile ducts, liver fibroses, and testicular degeneration.[3a]

Symptoms.—"This is an insidious chronic disease, with a course of several weeks to three months or more. One seldom has an opportunity to observe the initial stages. Owners reported watery discharge from the eyes and nose as the earliest changes seen. This was followed by a loss of condition, poor appetite, depression, and progressive thickening of the skin. The skin changes started over the withers, on the sides of the neck, over the cheeks and back of the shoulders. Finally, the upper two-thirds of the body was more or less involved. The hair was lost and the skin became dry, leathery, and deeply creased. Either this change caused little discomfort or the animal was too depressed to be disturbed. Itching or rubbing was not observed and the animal was not interested in licking or grooming itself. The skin changes sometimes extended down onto the brisket and occurred between the thighs in the region of the mammary gland. Except for this the skin over the ventral third of the body was not altered. No changes occurred on the feet and legs nor on the face. The salivation suggested oral changes, and inspection of the mouth often revealed papillary projections on the surfaces of the tongue, cheeks, or palate. . . . Nearly all the animals that showed marked skin lesions became emaciated, weak, and finally died."[7]

Other symptoms are red areas on the nostrils, lips and gums, intermittent diarrhea, reduced milk flow, and abortion. Often the epididymis is enlarged and hard. One of the most constant symptoms is a drop in the level of the vitamin A. It has been reported from Wisconsin[2] that more calves die from acute hyperkeratosis than live to develop the skin form, and there are various reports that calves which receive nothing but milk from an affected cow may acquire the disease.[2, 8] The most severe forms occur in the young and in the winter. In the differential diagnosis one may suspect either mange or malignant head catarrh. No effective remedy has been found.

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TRICHLORETHYLENE EXTRACTED SOYBEAN MEAL POISONING[1]

Definition.—Soybean oil meal poisoning is an acute hemorrhagic disease in cattle when fed certain batches of commercially prepared trichlorethylene-extracted soybean meal. Clinically there is a bloody discharge from the nostrils and in the feces. The essential cause is unknown.

Etiology.—The disease was first reported by Stockman[2] in England. There is an extensive literature on its prevalence in Europe and it has been observed in various states in the Midwest by Pritchard et al[3] who write that the young, the well-bred, and high milk producers are most susceptible, that the disease varies widely according to the toxicity, the amount fed, the number of highly susceptible young, and the number in advanced pregnancy. Where calves under six months of age were fed they were the first to have the disease, and it was produced in mature animals fed on 1 to 3 lbs. per head daily; calves fed ¼ to ½ lb. daily for five weeks died. Losses began 30 to 270 days after the start of feeding, most losses occurred within 30 days after the first death. Deaths might occur several months after the feeding was discontinued. The toxicity may be either low or high. In the herds described by Stockman from 1 to 19 per cent of susceptible animals in the herd were affected, with an average of 10 per cent.

Morbid Anatomy.—Autopsy reveals extensive hemorrhages throughout the body involving the gastrointestinal system, the subcutis, the respiratory system, the serous membranes, the kidneys, the liver, the

pericardium, the spleen, the bladder and the brain. The lymph glands and gall bladder are swollen.

Symptoms.—Cattle thrive for varying periods, 1 to 6 months, and symptoms appear suddenly only in the final stages of the disease, and at first in one or two animals. The onset is marked by depression, anorexia, a temperature of 106 to 108° F., chills, staring hair coat, suspended rumination and often salivation. The milk flow drops suddenly and blood may trickle from the nostrils or appear in the feces, yet in some herds visible hemorrhage may be absent. Blood may appear in the eyes, mouth, vulva, and prepuce, and noseblood may be profuse. Hemorrhage is the most characteristic symptom. Abdominal pain may be shown by kicking the abdomen and treading. Stiff gait may result from intramuscular hemorrhages. Hemoglobinuria is frequent. Deaths occur within four to five days after the onset.

In the *blood* the first indication is a marked and progressive reduction in blood platelets, early and progressive leukopenia is constant, appearing as early as two weeks after feeding begins and persisting as long as eleven months after it is discontinued. The hemoglobin is low and the blood-clotting time may be two hours compared with a normal of 10 to 15 minutes.

The history and symptoms differentiate it from sweet clover disease, hemorrhagic septicemia, and bracken fern poisoning which it closely resembles. There is no cure.

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FLUOROSIS

Definition.—Fluorosis of cattle and sheep is a chronic, insidious, and cummulative intoxication which interferes with cellular respiration through an attack on the enzyme system.[8, 10, 4, 23] It is caused by prolonged ingestion of fluorine and is characterized by stunted growth, intermittent lameness, mottling and irregular wear of the teeth, rough hair coat, low milk production and poor condition in the early stages, followed by permanent lameness, emaciation and difficulty in rising due

to osteoporosis. As the disease advances periosteal bone thickenings may appear on the mandibles, the ribs, the lower limbs and the digits. Because of osteoporosis the ribs may become brittle, and the fluorine content of the bone may exceed 5000 ppm or more, compared with a normal of 500 to 1000 ppm.[3]

Etiology.—Among the elements in the earth's crust fluorine is twentieth in frequency and it exists only in combination with other elements, as in bauxite, feldspar, cryolite, phosphatic limestone, etc. The nature of the contaminant which escapes in factory fumes is described as a solution of hydrofluoric acid (HF) in small drops of water carried in a mist and deposited on the green herbage where it is highly toxic,[11] and as a mist in which the effluents, presumably silicon fluoride (SiF_4), condense on the smoke particles that are not easily filterable by cotton wool and are readily dispersed over long distances,[3] and to direct contamination from the hydrofluoric acid that is emitted to the atmosphere through the manufacture of aluminum (MacIntire[27 p. 57]). Owing to the wide variations that may occur, it will be difficult to establish a reasonably safe tolerance level in forage that is contaminated with industrial effluents (Huffman[27 p. 60]). "It is not known definitely where or in what form fluorine is absorbed when a fluorine compound is taken orally."[1 p. 103] The normal values of fluorine in grass from unexposed areas is about 2 ppm in the dry matter, in the urine 2-10 ppm, and in bone 500-1000 (0.5-0.10 per cent).[3] Following suspension of exposure, changes in plants take place rapidly, those in the urine require months, and those in the bone require years. Fluorine in the soil is largely inert.[2]

Following the introduction of phosphorus supplements in the 1920s for livestock in the form of rock phosphate or phosphatic limestone, it was observed that "it caused abnormal abrasion of the teeth, affected the bones and also altered the general health as shown by the appetite."[7] It was soon learned that this condition was fluorosis caused by fluorine in the minerals.[6, 7]

The upper accepted limit of safety of fluorine for cattle and sheep is not more than .009 per cent of the grain ration. Since the prevention of fluorosis by ingestion of mineral compounds in the feed is well known and generally practiced, chief importance attaches to areas where industrial operations may contaminate the atmosphere, as from factories engaged in the manufacture of superphosphate, aluminum, bricks, steel, the calcining of ironstone, enameling processes, etc., and that use bauxite, cryolite feldspar or sodium fluoride for a flux. At least twenty-five occupations are possible sources of danger.[10] Unlike intoxication from mineral supplements, one may not know the nature of the compound, the amount and uniformity of distribution, the dosage, the

possible obscure and prolonged injury to livestock, or the possibility of its presence. Thus, farming operations which may be exposed to the hazard are under a cloud of suspicion which affects the sale of the animals and crops and even the farm itself. Increasing hazards from the industrial development of fluorine compounds have been expressed by Larner,[4] by Bond and Murray,[5] by Murray and Wilson,[10] who have described fluorosis affecting both animals and persons, and by the Fluorosis Committee.[12]

Pathology.—Except for advanced changes in the skeletal system (osteoporosis) and the teeth (hypoplasia) little has been written on the pathology of fluorosis. Other conditions are calcification of ligaments and tendons, and a "systemic reaction" which includes sterility, stunted growth, poor condition, lessened appetite, suppressed lactation, and the most common symptom of all—stiffness and lameness, which is often intermittent. In animals and man the joints are intact on both clinical and x-ray examination.[1, 3 p. 279, 10]

In an attempt to demonstrate the effect of fluorine hazard before such severe intoxication has resulted as to cause disability and obvious skeletal changes, Bond and Murray[5] observed that the effects on the kidney function are immediate. . . . Tubular function rather than that of glomeruli was affected, and tubular function depends largely on enzymic activity. And Kick et al.[24] wrote in 1935 "the addition of 1 per cent rock phosphate to the ration of pigs caused a degeneration of the epithelium of the convoluted tubules and a fibrosis of the kidney."

The postmortem findings in advanced fluorosis are described by Blakemore[3] and others,[1] as enlargement of the sternum, and inferior maxilla, periosteal thickenings of the limbs below the tarsus and carpus, on the digits and the upper third of ribs. The ribs are fragile and easily broken. The bones are more massive in appearance but of lower specific gravity than normal (osteoporosis). On maceration they are dull white and porous. The fluorine values range from five to 20 times the normal. The chief value of bone analysis results from an examination of bone tissue of several animals in a suspected area compared with corresponding checks from an unexposed area. Depending on the age, previous consumption of mineral supplements, and the presence of fluorine in the water,[1 p. 54] samples from individuals may greatly exceed the normal of 500 to 1000 ppm.[3]

Symptoms.—The complex and obscure nature of fluorosis is expressed in a statement by Roholm,[1] that "only gradually have the various symptoms of the chronic intoxication been recognized; but in many respects our knowledge is still defective," and, "The effect of

fluorine depends on the dose, the duration of ingestion, the age of the individual, the species, the composition of the diet, and probably other circumstances as yet unknown; only some of the various forms of intoxication are known." And by Phillips, Hart and Bohstedt[8] who write that "the changes in chronic fluorosis are a gradual transformation from the normal to unmistakable evidence of abnormal function; it is this insidious development which becomes of practical importance." The

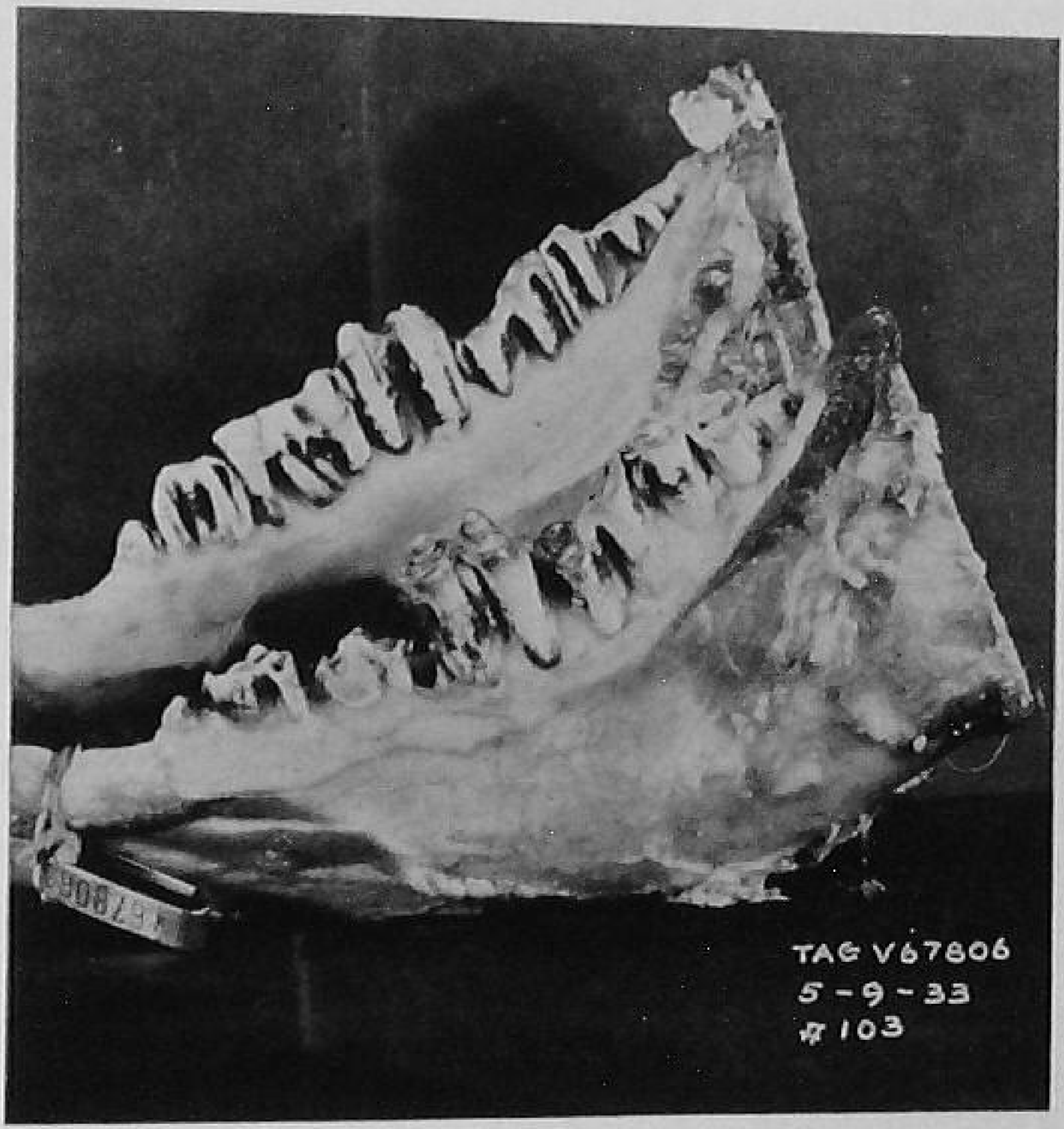

FIG. 110.—Atrophy of the teeth, caused by fluorine poisoning.

effect of fluorine is also influenced by wide variations in individual resistance, as in two animals raised together from calves, one of which grows normally while the other remains stunted and unthrifty. In exposed areas, farmers have learned that replacements from a distance are more apt to develop symptoms than animals purchased as apparently normal within the area. The young have a relatively low resistance and there is also a variation according to species. In rats growth and general condition begin to suffer at a daily intake of about 20 mg. per kg. body weight, while in cattle the tolerance is much less, about 2-3. mg. fluorine per kg. daily. Pigs and sheep are less sensitive than cattle and man is much more sensitive than the rat.

The Teeth: "Changes in the teeth are among the most easily reproducible and most easily recognizable of all the symptoms of chronic

fluorine poisoning."[1] In the mildest change there is a slight mottling of the enamel of the permanent incisors which might have been exposed to fluorine during eruption; they present chalky white spots of hypoplasia which become brown or black and pitted. The general discolor of the teeth is usually yellow, but it may be brownish or black. In a more advanced stage the incisors are shortened from abnormal wear due to softness, and the table surface is rounded. In cattle the anterior surface of the incisors is roughened from longitudinal ridges while a normal tooth is glossy white and smooth; the roughness of the anterior surface may be recognized by drawing a finger nail across the tooth. In advanced fluorosis the table surfaces of the cheek teeth show irregular wear which may become so marked that chewing is difficult (Fig. 110).

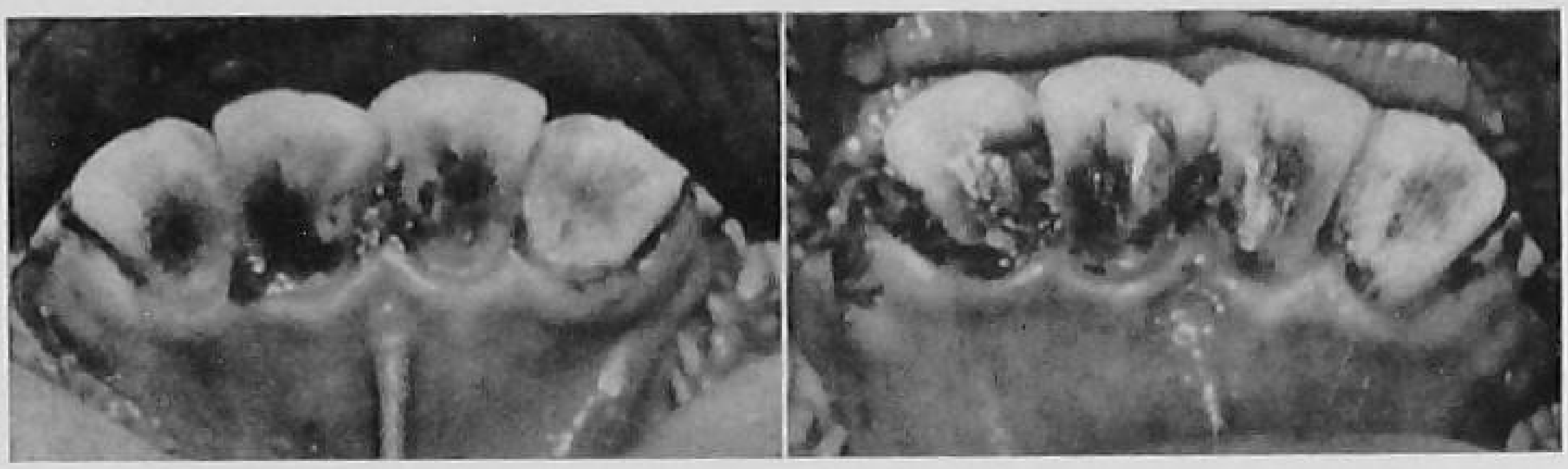

FIG. 111.—Extension of the staining of the teeth of one animal from May 30, 1950 *(left)* to October 26, 1950 *(right)*.

On farms where the cattle have been exposed to fluorine during eruption of the permanent teeth, and fluorosis is not advanced, the majority of the incisors show some degree of staining and roughness; a few may be apparently normal and a few may be highly stained (Fig. 111). In comparison with a neighboring herd equally exposed the number of animals with stained and roughened teeth may be greater or the teeth may present a more normal condition. In an advanced form of fluorosis the teeth are seriously involved. It has been shown experimentally that staining of the teeth of rats may result from a dosage of fluorine below that capable of producing any other visible symptom, and these observations have sometimes led to an opinion that absence of staining of the teeth and bony swellings preclude intoxication with fluorine, but in some cattle the teeth remain normal even when there has been exposure to fluorine during the period of their eruption. It is conceivable that the effect of a highly toxic form of fluorine may be systemic before a sufficient time has elapsed for changes to occur in either the teeth or the bones. And it is a common observation that animals vary widely in their reaction to fluorine. When animals whose permanent teeth are in

process of eruption are moved from an unexposed to an exposed area, the teeth which erupted in the unexposed area are normal, while the others are stained; this distinction has been observed repeatedly. Because of painful gums a heifer with permanent teeth in the process of eruption may drool and lap water. Exposure to fluorine during the eruption of the permanent teeth, between the ages of two to five years, results in staining and defective development, of the permanent teeth, and these defects are pathognomonic of fluorosis. The temporary teeth are rarely affected, and if there is no exposure until after complete maturity of the permanent teeth, at about five years of age, these teeth remain normal regardless of subsequent exposure. A herd is probably

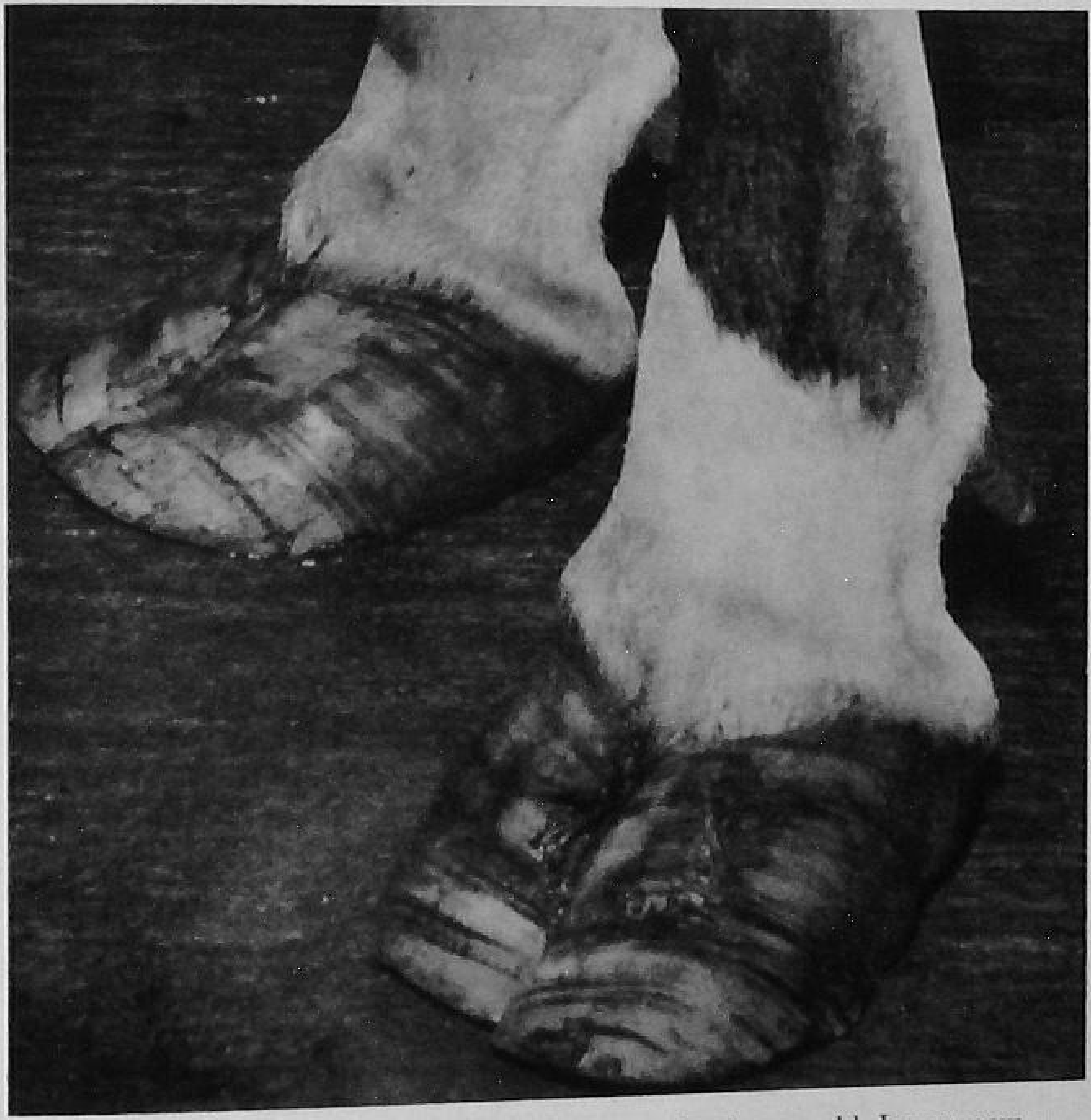

Fig. 112.—Condition of the front hooves of a 7-year-old Jersey cow after three years in herd.

free from exposure when the incisors of all the individuals are glossy, smooth, and white.

Lameness.—Aside from lameness and bony swellings in an advanced form of fluorosis, lameness from which cattle and sheep recover is one of

the most frequent symptoms. In affected herds one often hears the statement that nearly all have been lame at one time or another. In common with the symptoms of fluorosis in general the lameness varies widely in different herds and at different times in the same herd and more are affected than are found to be lame at any one time. It occurs as a permanent stiffness, a recurrent lameness at each parturition, a

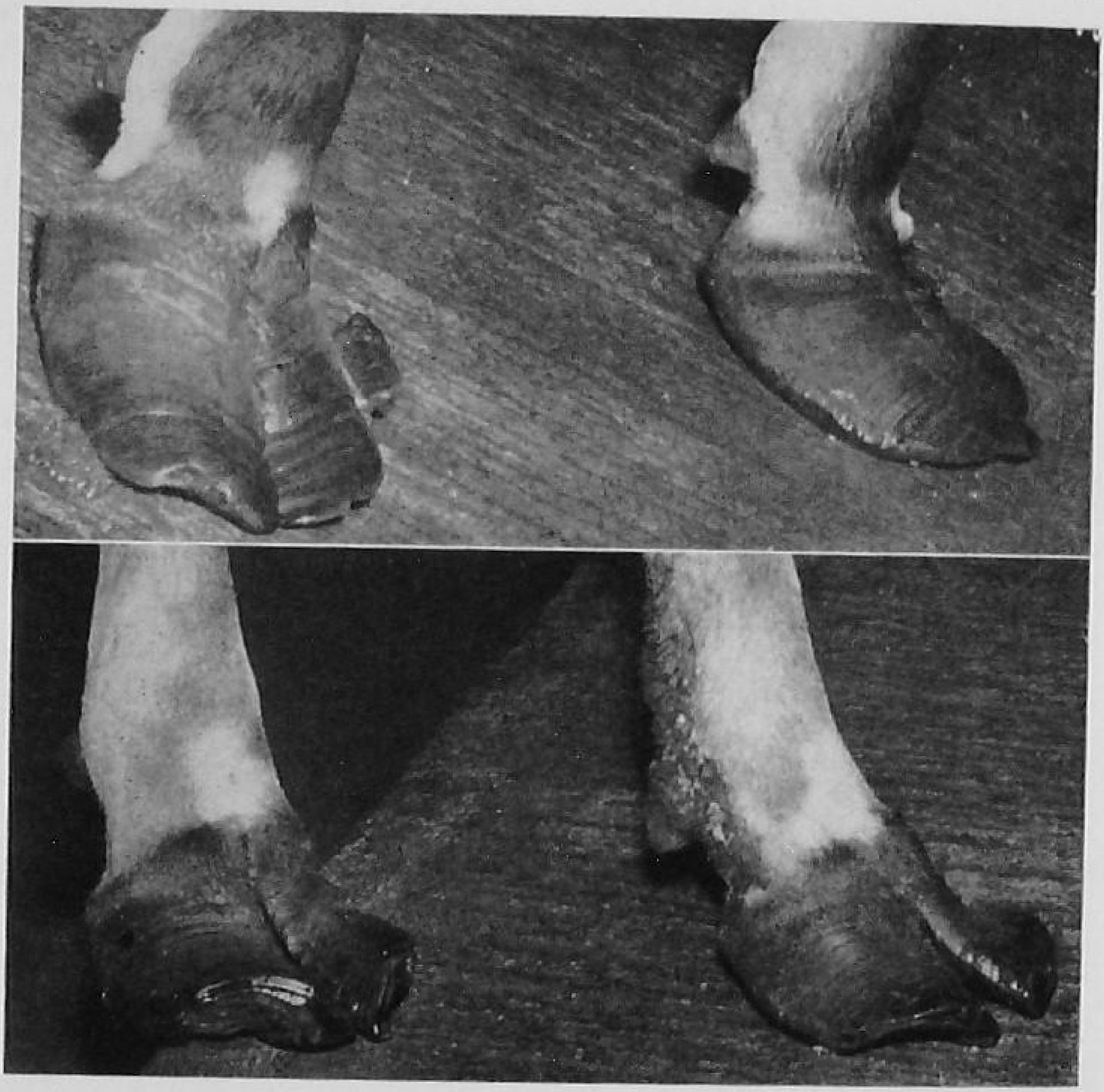

FIG. 113.—Condition of the hooves of a 4½-year-old Jersey cow after three years in herd. *Above:* front hooves. *Below:* rear hooves.

crippled gait, an arched back, and overgrown deformed hoofs (Fig. 113). Slagsvold[25] writes that the animals remain down much of the time, rise with difficulty, are sore-footed and have a stiff gait, yet the joints and tendons may be apparently normal. Bosworth et al.[26] state that "Within a few weeks after being turned out to grass on affected farms the animals showed signs of unthriftiness, and some looseness of the bowels and soon developed a stunted appearance. Within three or four months signs of lameness appeared and gradually became more marked.

When the cattle were housed and fed on partly concentrated food they showed some improvement, but soon became worse on returning to pasture." After removal of lame cows from grazing approximately a month is required for recovery.[21] There is no explanation for the nature of the pathology when the lameness is transient, and radiographs of lameness in human patients have been negative.[10]

Occasionally a snapping sound may be heard in the feet of cattle when walking and this has been attributed to separation under pressure of overgrown and crossed claws. A similar sound in the ankles of a human case was attributed to calcification of the ligaments and tendons and it is possible that the same explanation applies to the snapping

Fig. 114.—Condtion of the rear hooves of a 9-year-old Holstein cow.

sound in the feet of cattle. In cattle, in the early stages, the hoof may flatten out to form an acute angle with the floor, sometimes termed "shovel-hoof or duck-hoof." In one such animal observed two years later the hooves resembled those of a horse with chronic laminitis, the lameness was still severe, yet the milk production was reported as always good, and the general condition had improved. A letter from P. L. Shanks, M.R.C.V.S., who investigated fluorosis in Yorkshire, England, where lameness was a main feature, states that animals with hooves similar to those in Fig. 112 had fractures of the os pedis, either fresh or healed. This deformity of the hooves has also been observed by Fincher[14] in cows in Massena, N.Y., and Slagsvold[25] mentions the special tendency in cattle to overgrowth of the hooves in fluorosis, even on stony pastures where normally they are worn down. Figure 9 shows the rear hooves of a 9-year-old cow that could hardly walk because of stiffness and pain; when walking the weight was borne on the

heels and the toes were tipped up. Another form of lameness begins as a diffuse swelling centered about the carpal joint and involves a large part of the limb; the lameness as well as the swelling may be intermittent, and may eventually recede until only a bony circumscribed swelling remains just below the fetlock joint on the side of the first phalanx, or there may be a bony circular swelling just above the hoof.

Growth.—All authors who mention growth report that it is retarded by fluorine.[15] This applies especially to those who have encountered fluorosis as it is found on the farm in both cattle and sheep; thus, in the report by Blakemore et al.[3] one reads that "evidence regarding the specific effect of fluorine intoxication on the general health of the animal is difficult to assess, but the growth of the young stock was retarded." A group of calves turned to pasture on a contaminated field when weaned at the age of four months may be stunted as yearlings, too small to breed at the usual age, and a year late at the time of the first parturition, while a corresponding group from the same farm may make a normal growth when placed at pasture in an adjoining county. When there has been excessive feeding of contaminated mineral supplement, and in areas exposed to aerial contamination, a herd examination may reveal one or several adults that have failed to reach the size of their older associates that were not exposed during growth. In advanced fluorosis caused by feeding phosphatic limestone, "yearlings and two-year-olds which were together in the same pen were all of the same size; mature cows were no larger than two-year-olds."[13] In the notes by Fincher[14] on cattle in the Massena, New York, area, "there was a distinct loss of condition in all animals, with emaciation in several. The heads appeared large in some of the younger animals, and their bodies were definitely underdeveloped (stunted). There were about three normal sized cows in the herd of 30 adult animals." "Successive generations of rats fed very small amounts of fluorine, 0.025 per cent, apparently produce well, but do not grow normally."[17 p. 35]

Reproduction.—Sterility in cows has been observed repeatedly and it may vary in dairy herds from a low percentage to one in which nearly all replacements are by purchase; in other herds reproduction may be normal. Among the reported effects are atrophy of the testes and absence of sperm in rats fed a supplement of 0.15 per cent (1500 ppm) sodium fluoride;[9] lowered weight of newborn calves and delayed postpartum estrum in cows fed 0.0625 per cent rock phosphate;[8] impaired reproduction and retarded growth in rats fed a supplement of 0.10 per cent sodium fluoride;[18] successive reproduction is usually slow in rats that have been raised through seven generations on small amounts of sodium fluoride;[17] in rats, systemic reaction suppresses re-

production and lactation;[16] an unfavorable effect on reproduction;[1] estrum every nine days in cows on 2 per cent phosphatic limestone for four years;[13] and abortions and weak calves in cows fed a grain supplement of 3 per cent rock phosphate.[7]

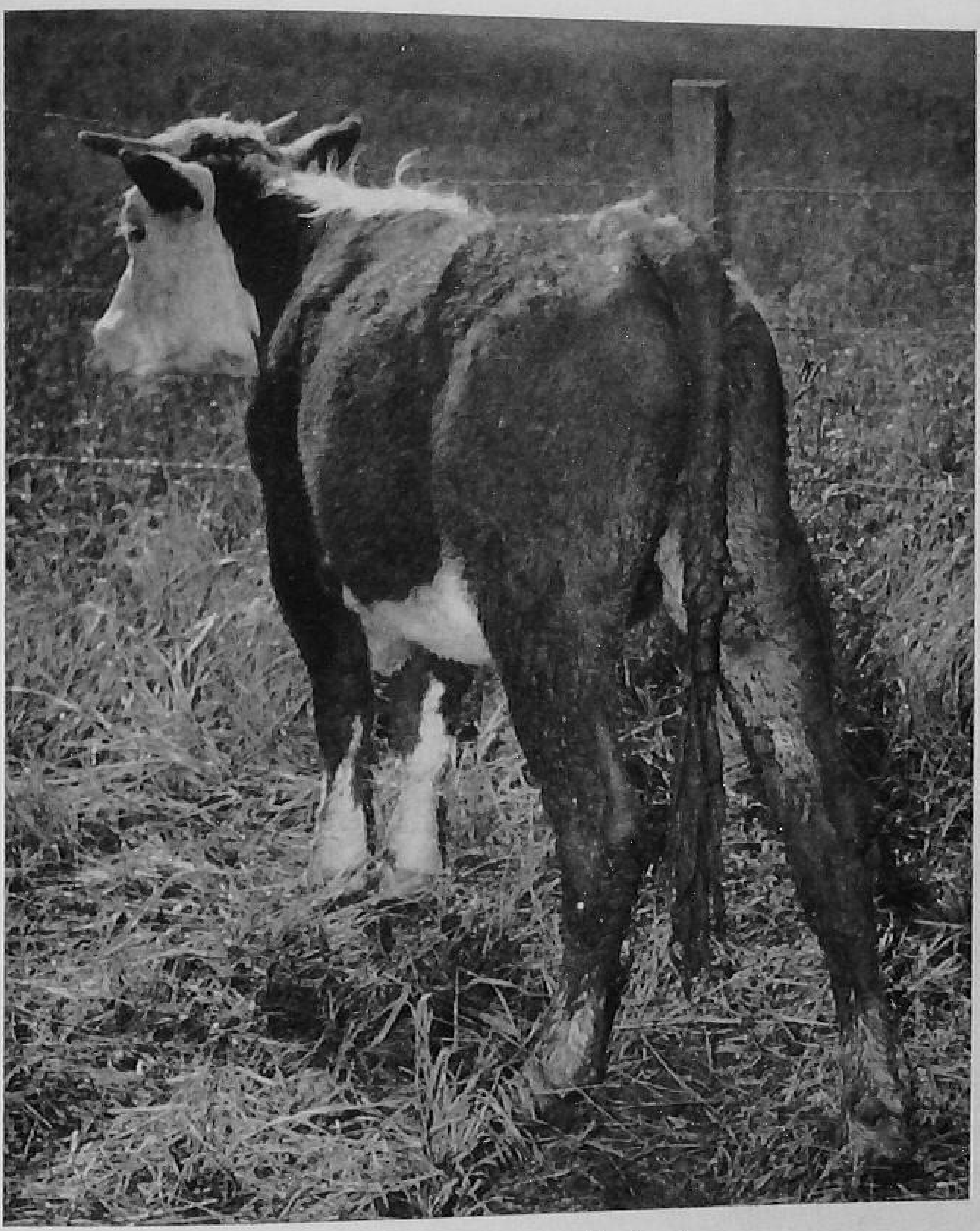

FIG. 115.—Stunted calf with diarrhea.

Condition.—"Emaciation is a marked feature of all farm animals suffering from chronic fluorosis. . . . The values we have obtained for urinary nitrogen excretion show that the emaciation could in part be due to an increased metabolism (total nitrogen 44-56, increased to 120-134 mg) 180 hour collections."[5] "*Milk suppression* is a result of systemic reaction to fluorine toxicosis."[8]

Digestive System.—In the early recorded observations on fluorosis from feeding mineral supplements one finds a report of altered general health and diminished appetite.[7] Within a few weeks after being turned out to grass on affected farms the animals showed signs of unthriftiness and some looseness of the bowels.[26, 12] "The cows often have a soft discharge or even diarrhea, they develop anorexia, lose weight and give little milk."[25] Difficult chewing appears in the advanced form with deformity of the molars. Diarrhea in young stock was a prominent symptom in one herd in Oregon,[20] (Fig. 115) the faeces were black, tarry, and tenacious and were thickly smeared over the buttocks, the entire length of the tail, and the hind limbs. The owner reported that with a spell of clear weather there was an improvement in the diarrhea which returned with rain or fog. Similar observations were reported by others, and veterinarians reported failure in the treatment of diarrhea in animals in this area.

Urinary System.—"Abundant liquid intake and polyuria is a common symptom in several species of animals in experimental intoxication."[1 p. 271] Under field conditions these symptoms are difficult to recognize in large animals, but in one large herd in Oregon they were apparently present.[20] And Blakemore et al.[3 p. 279] write that "diuresis and excessive thirst, which have frequently been recorded as symptoms of fluorosis, were not observed and there was no diarrhea.

Urine.—The value of urine analysis for the determination of fluorine has been reported by Blakemore et al.,[3] who found that "the most practical method of assessing the degree of fluorosis was to determine the current rate of fluorine excretion in samples of urine, easily obtained by catheter and promptly analyzed by appropriate technique. This method is not only useful in substantiating a clinical diagnosis, but it is also invaluable in plotting the extent of areas of abnormal fluorine ingestion and the severity of effects. . . . In advanced clinically detectable fluorosis urinary values varied from 16 ppm to 68 ppm with a preponderance of values well over 25 ppm as compared with normal values of 2 to 6 ppm. . . . It would, therefore, seem that a urinary value of 10 ppm is somewhat critical from the diagnostic point of view."

Diagnosis.—"The symptoms in advanced fluorosis are sufficiently characteristic for diagnosis, and when dental changes are present the disease is not likely to be confused with any other condition."[3 p. 270] In the more frequent earlier stages, and especially in factory areas, one needs to consider the entire herd, the distance from possible sources of exposure, the direction of prevailing winds, the rate of reproduction, the age of the animals, the rate of replacements and the reasons for sale or disposal of stock, the provision for adequate minerals (Ca and

P), and the history of the herd and of neighboring herds. While analyses of the urine and bone and forage are desirable, under usual field conditions they are difficult to obtain, and the interpretation of results of a single analysis may be difficult. While Phillips writes that in cattle bone may acquire 4000 ppm without causing injury, Blakemore[3] reports a rib analysis of 5900 ppm when the ribs were so brittle that they could be broken by hand after removal from the body. And Boddie[21] reports in affected sheep a mandible analysis of 4200 ppm and 2140 ppm with a normal of 415 ppm, and in affected cattle, a rib analysis of 3220 ppm with a normal of 114.2 ppm. As expressed by Blackemore et al.,[3 p. 271] "For values of 'bone ash' (the conventional mode of expression) fluorine values of 0.05 per cent to 0.10 per cent (500 to 1000 ppm) are regarded as quite normal, but can be greatly exceeded before fluorosis is clinically detectable."

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BRACKEN POISONING

(Bracken)

Etiology.—Bracken poisoning is an acute, sometimes subacute, intoxication in cattle caused by grazing on ferns *(Pteris aquilina)*. It is characterized clinically by a sudden onset, high fever, and hemorrhages in the mucous membranes and skin; pathologically it is marked by extensive hemorrhages, especially in the abomasum and intestines, and by necrotic foci in the liver. Apparently this disease was first described by Storrar[1] in England under the title of *Cases of Vegetable Poisoning in Cattle*, which he attributed to grazing on young fronds of the bracken fern. The highly characteristic symptoms and lesions were described by Stockman[2] in 1917, who reported that symptoms and lesions in cattle poisoned by soy bean meal which had been defatted with trichlorethylene were identical with those caused by bracken poisoning, and that he produced bracken poisoning by feeding bracken shoots to an 8-months-old animal; the total amount consumed was approximately 260 pounds. He considers it probable that the toxic principle is the same as that present in soy bean meal, possibly of the nature of ricin, abrin, etc. Hadwen[3] observed bracken poisoning in horses in the Frazer River Valley. It developed in the months of January and February in horses fed exclusively on hay containing bracken *(P. aquilina)*. Wetter[4] has also described bracken poisoning in horses in the winter months when fed upon hay containing large amounts of fern. According to his experience only certain meadows seemed to harbor the toxic fern, and this action was more marked after a dry hot season. The symptoms were uncertain gait, loss of equilibrium, and poor condition. When neglected

the horse may develop marked nervousness, paralysis, and inability to rise. The disease of horses, described by Hadwen and Wetter, bears little resemblance to bracken poisoning as it occurs in pastured cattle, but it does resemble poisoning by *Equisetum* (Horsetail). "Animals will develop symptoms two or more weeks after removal from ferns."[5]

In New York State the disease occurs chiefly in the months of August and September in pastures that contain little natural forage. Thus it is more prevalent when the fields have been overstocked, and when the summer has been unusually dry. After the first frost the disease terminates abruptly. It is highly prevalent only in certain years. Usually only one or two animals in a herd are attacked, but there are frequent exceptions. Yearlings and two-year-olds suffer chiefly, yet it is not uncommon in mature cows when the season is excessively dry. In this State the disease was first described by Bosshart and Hagan.[7] The following year Hagan and Zeissig produced the disease experimentally by feeding brakes *(Pteridium latinusculum)*, formerly called *P. aquilina*.

Morbid Anatomy.—Examination of the cadaver may reveal evidence of intense exertion or convulsions at the time of death, as shown by patches of bare turf in the immediate vicinity. Also the ground may be covered rather extensively with streaks of bright red blood which came from the rectum. There may be froth at the mouth and nose, and a blood-stained skin. On opening the cadaver nearly all of the tissues may show hemorrhages; these are present in the skin, subcutis, intermuscular tissue, peritoneum, bladder, lymph glands, kidneys, and even in the fetus.[6] The *abomasum* is edematous and hemorrhagic, especially at the duodenal opening; the hemorrhages may be recent or several days old and rest upon an area of ulceration. In the *small intestine* the lesions are like those in the abomasum. The *cecum* and *large intestine* are often filled with a large mass of bright red clotted blood. The *liver* usually contains necrotic areas scattered throughout the entire organ; these may be microscopic in size or attain a diameter of an inch. They closely resemble the necrotic foci caused by *Actinomyces necrophorus*. The *lungs* sometimes contain foci similar in appearance to those in the liver. The mucosa of the air passages and nasal sinuses may contain ulcers. In the *kidneys* there may be hemorrhagic nephritis, but this condition is not the rule. The *serous* and *mucous membranes* are thickly covered with ecchymoses. The *spleen* is usually normal.

Symptoms.—As a rule the onset is sudden with an extremely high fever, the temperature being 106° to 109°F. The animal stands with the head lowered, drooling saliva, and bloody fluid may trickle from the nostrils. The visible mucous membranes nearly always show petechiae; these may be observed on the vagina, in the nasal passages, and on the

lips, particularly the lower lip below the incisors.[7] In the male, hemorrhages may be found in the scrotum. Dyspnœa is occasional, and hematuria may be present. The course of the acute form is from one to three days, nearly always ending in death. A *subacute or chronic form* which lasts from four to ten days has been described. The chief characteristics are anorexia, slight nosebleed, necrosis of the base of tongue, pharynx, nostrils, and trachea, and areas of necrosis in the skin of the hips, shoulders, prepuce, fetlocks, and muzzle; these resemble lesions caused by *A. necrophorus*. Jaundice is constant and increases with the progress of the disease. In some cases large clots of blood are passed from the bowel. The course is from four to ten days and only a few recover. The changes in the blood are leukopenia with reduced numbers of polymorphonuclear leucocytes and blood platelets.[5]

The *diagnosis* is important because of the resemblance to acute general infections. It has frequently been mistaken for hemorrhagic septicemia, and it may be confused with either anthrax or blackleg. No effective remedy has been found.

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EQUISETUM POISONING

Etiology.—Equisetum poisoning is an acute paresis or paralysis affecting chiefly horses, and caused by feeding hay heavily contaminated with *Equisetum arvense* (horsetail fern, foxtail, scouring rush) which is native throughout the United States and Canada.

An unidentified toxic substance exerts a paralytic action on the spinal cord and cerebellum, and Fröhner[1] writes that depending on the climate and locality the plant may be toxic or nontoxic.

This disease was described by Rich and Jones[2] in Vermont in 1902, where Equisetum is more or less prevalent on many farms in the State, and a frequent cause of fatal poisoning; in the vicinity of Burlington, Dr. Rich observed 23 cases in a period of two years. Equisetum thrives in low, moist, uncultivated sandy meadows subject to overflow of

streams, where it is often associated with polypod brake (*Onoclea sensibilis*—sensitive fern). It is said to be toxic to sheep and nontoxic to cows. Crane[3] has reported from England the poisoning of cows fed on hay containing one-third *Equisetum palustre*, while Rich mentions one farmer whose cows apparently thrived on hay containing 25 per cent *Equisetum arvense*. Opinions on its toxicity for sheep are conflicting.

Symptoms.—The first symptoms are often in the form of an increased irritability, as shown by hypersensitiveness to sounds and movements; the animal is nervous and easily startled, showing fear, excitement, or anxiety. Thus Dr. Rich, in describing various cases, states that the animal was nervous and startled at slight noises, or trembled from fear when approached, or developed profound fear of bicycles and electric cars, or became nervous and began to reel in walking, or when approached or handled became frightened and attempted to run away. The horse is at first unthrifty and thin, and in two to five days, according to the age and manner of feeding, it staggers when walking, avoids lying down, finally falls down when no longer able to stand, and struggles violently to rise. Some have likened the reeling and staggering movements to those of a drunken man, or to an animal on slippery ice, and it is known in Germany as the "tumbling disease." Until the animal goes down there are no general symptoms, such as depression, inappetence, increased pulse, respiration and temperature, with the possible exception of a subnormal temperature and a slow pulse. There is no jaundice or anemia and the mucous membranes are normal. In a condition of extreme weakness, and a tendency to stagger there may be a ravenous appetite. Dr. Rich writes that the eye is bright, the animal eats well and may caper and play, even when partially paralyzed. After falling, the legs become more or less rigid, at times all of the muscles of the body seem to be convlused; and in this condition the victim may survive for two weeks before death occurs from exhaustion. The old are less susceptible than the young; on one farm a mare came down after four weeks and the colt by her side developed the disease and died in ten days. A depraved appetite for Equisetum may be acquired, when the horse leaves grain to eat bedding heavily contaminated with horsetail. Constipation is usually present. An increased resistance is found in grain-fed horses. As a rule the course is several days, and Fröhner[1] writes that it may terminate fatally in a few hours. A chronic form has been described in young animals that receive less than the fatal amounts; these animals are unthrifty, unsteady, nervous, and easily startled.

The *diagnosis* is not difficult unless one fails to correlate the symptoms with the cause. It is probable that many cases of foxtail poisoning

have been diagnosed as botulism, "food poisoning," encephalitis, and paralysis of unknown cause, and it is reported that in northern New York it is frequently confused with infectious anemia.

On *autopsy* the muscles are pale, while the meninges of the cerebellum and spinal cord are congested. In one case there was a serous turbid exudate around the cerebellum (Rich). Comparatively little has been written on the pathology of this disease.

Treatment.—The prognosis is good if the hay is changed before the animal goes down, and recovery may sometimes occur even after the horse is unable to stand, if its position is frequent changed and slings are used for support. A laxative of aloin, and strychnine sulfate is recommended by Rich, while others limit the treatment to change of hay and care of the patient.

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INFECTIOUS KERATITIS

(Infectious Conjunctivitis; Pinkeye; Specific Ophthalmia)

Definition.—An infectious and contagious acute conjunctivitis and keratitis caused by *Hemophilus (Moraxella) bovis.*

Etiology.—In 1923 Jones and Little[1] reported on an outbreak of ophthalmia that was introduced into a dairy herd in New Jersey by cows recently purchased from a dealer in Ohio. From all 24 cases observed a characteristic diplococcus, *Hemophilus (Moraxella) bovis* was recovered, and "instillation of a few drops of bouillon suspension

FIG. 116.—Infectious Keratitis.

of pure culture beneath the eyelids of normal cattle gave rise to characteristic inflammation." In 1945 Reid and Anigstein[2] reported that "the disease is highly contagious among cattle, and can be readily transmitted in calves by contact with the eye or nasal exudates. . . . Systematic investigation of the bacterial flora of infectious keratoconjunctivitis revealed, in addition to various coccal organisms cultivated on ordinary media, a hemophylic and hemolytic bacillus which requires blood or serum for growth. Experimentally keratoconjunctivitis in cattle, sheep, and goats was produced by young cultures of the bacillus.

In addition, agglutinins specific for this organism were found in the sera of cattle convalescent from the natural or experimental infection. It is concluded that this hemolytic diplobacillus is the specific causal agent in cattle. Morphological, as well as cultural and pathogenic characteristics indicated close relationship if not identity with *Hemophilus bovis*." Baldwin[3] found *Hemophilus bovis* in 93 of 112 infected eyes examined and in no eye of 20 normal animals, and the infection persisted for two or more months after apparently complete recovery. And Farley[4] writes that in the chronic form, panophthalmia, infection may be carried through the winter and reinfect calves in the spring. In the United States infectious keratitis is widely prevalent among range and feed-lot cattle, where it may prevail to some extent the year round, and the number affected at any one time may be so few that its contagious features are hardly recognized. It is frequent in all parts of North America; it has been reported from Africa and India, and it appears to be world-wide. When an infected animal enters a herd in the eastern part of the United States in the fall, while cattle are at pasture, it soon spreads over the entire neighborhood. It is most prevalent as a pasture disease in the late summer and fall, but it has been observed in the winter in stabled animals in New York and Ohio. From 40 to 50 per cent of a herd may be affected, and it is most prevalent and severe in the young stock. Flies have been suspected as carriers of the infection, and Farley[4] observed that it was not transmitted to susceptible calves together in fly-proof stalls, even when they ate and drank from the same receptacles; that experimental infection of one eye did not involve the other; and that the lachrymal secretions proved virulent four months after the onset of the disease.

Symptoms.—The incubation period in experimental animals is from two to four days. The onset is sudden with photophobia, lachrymation, and swelling and redness of the lids which remain closed and are painful on palpation. There is a prominent matting of the hair of the eyelids and the face, and dermatitis with sloughing has been observed. After two or three days, if the lids are forced open, one finds a yellow deposit over the entire surface of the cornea, and viewed from the side there may be a cone-shaped bulging of the cornea caused by intraocular pressure. At the point of this bulge an ulcer may form, and in severe attacks it is the seat of perforation of the cornea with subsequent infection, involvement of the eyeball, and permanent loss of sight. As a rule, however, the corneal opacity finally disappears and the sight returns. The most severe forms are found in range cattle, where the afflicted are exposed to sun, dust, flies, and wind. It is said to be most severe in

white-faced cattle. The course is from two to four weeks with a high percentage of complete recovery. In a mild form there is lachrymation with a transient whitish or smoky turbidity of the cornea. There may be depression, rarely fever, and cows shrink as much as 50 per cent in the milk flow. Any severe attack in a dairy herd causes heavy loss.

Treatment.—Early vaccination with a bacterin containing *Hemophilus bovis* is said to prevent spread of infection in a herd after the disease has already appeared; the dosage is 15 cc. In western Kentucky, where keratoconjunctivitis in cattle is widely prevalent, Rose[5] has reported on the use of a "newer mixed bacterin" in 38 herds. Of these, 21 remained uninfected, and in 10 herds the infection was less than 1 per cent. Rose gave three weekly injections of 5, 10, and 15 cc. early in the spring. In a Texas report, efforts to immunize 38 Hereford calves is described; 12 developed complete immunity, 12 developed a slight conjunctivitis, whereas 14 head showed no evidence of immunity. For local treatment of the eyes sulfathiazole or penicillin ointment is beneficial. Bardens[6] gives credit to mixed bacterins combined with mercurochrome; the initial dose of bacterin was 10 cc. daily until a maximum of 20 cc. was reached. He found that two doses were usually sufficient in dairy cattle. Chloromycetin ointment, 200 mg. of chloromycetin in 3.8 Gm. of ointment is reported by Riley et al.[10] to be highly effective in the treatment of pinkeye in both cattle and sheep.

Infectious Keratitis in Sheep has been described by Coles,[7] Johnson,[8] and others.[9] As described, the symptoms are very similar to those of keratitis in cattle. The infective agent is said to be *Rickettsia conjunctivae*.[11] Johnson writes that it presents the appearance of trachoma.

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DISPLACEMENT OF THE ABOMASUM IN CATTLE

A report in 1950 by Begg[1] mentions briefly three cases of displacement of the abomasum in one of which the dislocation was found on laparotomy of a cow showing atypical symptoms of traumatic disturbance. Replacement was effected after removal of a considerable volume of the ruminal contents, but the manipulative interference caused a chronic peritonitis and the case gradually deteriorated. Two other cases showed inappetence, a decrease in the passage of feces and tympany at the border of the last rib on the left side; these were given the same diagnosis and withholding of the food for two days resulted in a return to normal.

This was followed in 1952 by a report by Jones[2] of the Department of Veterinary Surgery, University of Bristol, England, who also reported three cases. The first was found during a rumenotomy for the relief of traumatic reticulitis in a cow, and was corrected by manual manipulation of the abomasum through the incision in the left flank. The second case, in a three-month-old Jersey calf, was observed during an "exploratory laparotomy" following chronic abdominal tympany which could not be relieved by stomach tube or flank puncture. Manual correction was not attempted. The third case was observed in a seven-year-old Jersey cow fourteen days after her fourth parturition. The observed symptoms were inappetence, signs of abdominal pain, and a decrease in the amount of normal feces. Temporary improvement followed the administration of liquid petrolatum followed in a few days with recurrent inappetence, rapid loss in condition, reduction of milk yield, and suspension of ruminal contractions. Acetone appeared in the urine. Laparotomy was performed during the fifth week postpartum. Exploration of the abdominal cavity revealed an empty rumen, slight peritoneal exudate, and an ovoid discus distended with fluid and gas approximately 12 inches long by 8 inches across and related to the reticulum and left lateral wall of the anterior blind sac of the rumen; this was concluded to be the abomasum. The position was corrected by means of upward thrusts on the ventral surface of the omasum, and recovery was rapid.

The two previous reports indicate the presence of displacement of the abomasum as revealed by laparotomy, usually without previous sus-

picion of the primary disorder, or final comprehension of its significance. Attacks of persistent acetonemia, leading to disposal of the animal as worthless, have been a common experience in cattle practice. Recently, Moore and associates[3] of the Department of Surgery and Medicine, Michigan State Veterinary College, have presented data from the observation of thirty-three cases of displacement of the bovine abomasum which is of great importance in the control of diseases of dairy cows. Apparently they have solved the problem of the persistent incurable attack of ketosis by showing that the ketosis is secondary to a primary displacement of the abomasum. It is highly significant that 31 of 33 cases were associated with the late stages of pregnancy, or began within two weeks following parturition, and the other two followed immediately after postpartum paresis, and that at least half had previously been treated for acetonemia by practitioners without success. In the one complete description by Jones[2] the symptoms became obvious fourteen days after the fourth parturition. The discussion by Moore et al[3] shows that while the etiology is not clear, in eleven instances the pregnant uterus had descended under the rumen instead of being deflected to the right, as revealed by exploration of the abdomen during operation for relief. This would tend to elevate the rumen, to push the abomasum forward, and cause it to double up. Because of its anatomical attachments the central part along the great curvature is free to undergo considerable variation in position and it was quite apparent that the latter was the portion that slipped under the rumen and then dorsally between the left rumen and the abdominal walls. In all cases that part of the abomasum thus impounded had become greatly enlarged by distension by gas and liquid ingesta. In a few cases it was nearly as large as the rumen; in all it appeared to be at least double in size.

They write that we can now diagnose these cases with reasonable accuracy. There is a sudden decrease in the appetite which soon becomes intermittent. The most constant symptom is ketonuria. The feces are scanty, soft and pasty. On rectal examination the rumen seems to be located in the middle of the right abdominal segment instead of the left. The animal is dull, looses rapidly in condition, and the milk flow is greatly diminished. The temperature remains normal and the differential blood count is within the normal range. Available observations indicate that affected animals do not die, but finally become emaciated and are destroyed as worthless.

Under proper treatment the prognosis is favorable. Laparotomy in the left flank reveals the abomasum as a tubular structure 6 to 8 inches or more in diameter curving upward and forward just anterior to the last rib. Treatment employed successfully at the Michigan State College

consists in reduction of the size of the abomasum by release of gas through a 12-gauge needle attached to a length of rubber tubing to carry the gas out through the incision. Two umbilical tape sutures, 18 inches or more in length, are anchored to the wall of the rumen as far below the 7-inch incision as possible and the ends brought out to the exterior. If a steady pull is exerted on the tape sutures, the rumen can be rotated clock-wise from its normal position through about a 45-degree arc. Before starting rotation, the operator pushes the abomasum down as far under the rumen as possible and holds it there with his hand and forearm while an assistant relaxes the tapes; as the rumen rotates back to its normal position, it sweeps the abomasum along with it. Usually several attempts are necessary before complete reduction is effected.

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NORMAL RANGES BLOOD*

SPECIES	WBC $\times 10^3$	Segmented Neutrophils	Band Neutro-phils	Eosinophils	Basophils	Monocytes	Lymphocytes	RBC $\times 10^6$	Hemo-globin g/100 ml.
Cattle	4.5–13	30% (10–40%)	0–4%	8% (1–15%)	0–1%	9% (3–15%)	52% (40–80%)	5.4–9	8–14–5
Horse draft	5–11	56% (50–65%)	0–4%	4% (1–5%)	0.5 (0–1%)	8% (2–12%)	30% (20–40%)	6.5– 9.4	9–14
Horse Thorough-bred	8–15	45–60%	0.4%	1–5%	0.5%	6% (1–8%)	46% (35–60%)	8–13	11–17
Sheep	4–12	40% (20–50%)	0–4%	6% (0–15%)	0.2 (0–2%)	4% (1–12%)	52% (40–70%)	8.5–13.5	9–14.5
Goat	5–13	36%	0–4%	3.5%	0%	2%	58%	12.5–22	9–14
Swine	8.6–20	39% (30–50%)	0–4%	4.5 (1–10%)	1% (0–4%)	3% (1–10%)	52% (40–60%)	5.9	9–16.8

WBC expressed in thousands, RBC expressed in millions.
Figures in parentheses indicate ranges. The other figures are mean values.

NORMAL RANGE OF CHEMICAL CONSTITUENTS

SPECIES	Blood in mg/100 ml.				Serum in mg/100 ml.		
	Glucose	Total Non-Protein nitrogen	Urea Nitrogen	Total Ketones	Calcium	Inorganic Phosphorus	Magnesium
Cattle	40–60	20–40	6–27	Up to 5	9–12	2.3–9.6	1.8–3.1
Horse	60–110	20–40	10–20		9–15	2.4–4	2–3
Sheep	40–65	20–45	8–20	Up to 5	9–12	2.5–9	2.6
Goat	43–65	30–44	13–28		9–12	3–11	
Swine	40–125	20–45	8–24		9–15	4–11	

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* Compiled by John Bentinck-Smith.

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